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March 1991

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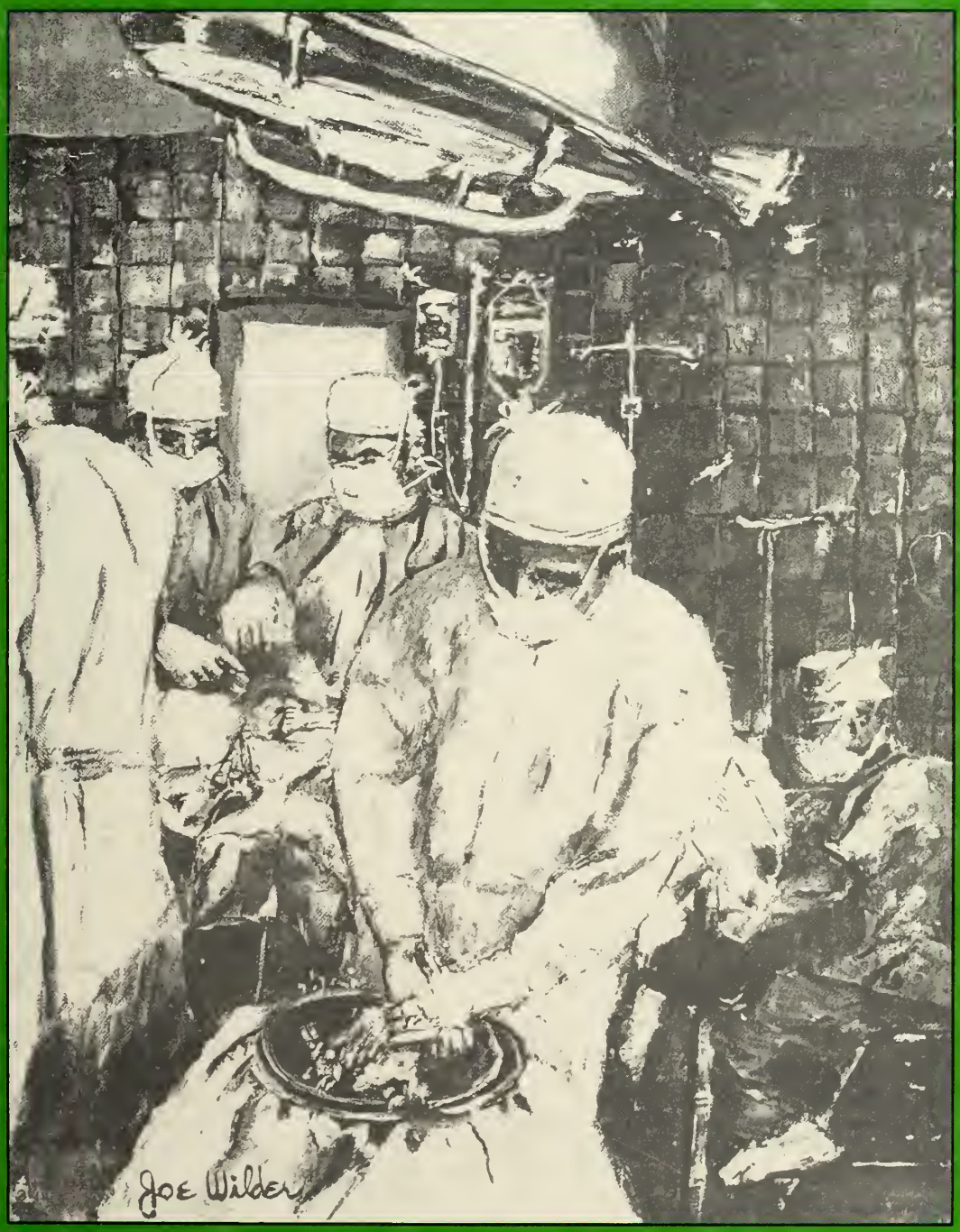

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Volume 74, Number 2 February 1991

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TABLE OF CONTENTS

99 **EDITORIALS**
The Art of Joe Wilder, MD
Should Practicing Physicians Be Routinely Tested for the AIDS Virus?
The Providence Medical Journal

CONTRIBUTIONS

105 **Deciding for Others: Surrogate Decision Making for Incompetent Adults**
Dan W. Brock, PhD

113 **Acquired Immune Deficiency Syndrome and the Surgeon**
Steven Schechter, MD
Joseph Amaral, MD

117 **The Clinical Use of Digibind in Digitalis Toxicity**
Richard V. Morgera, MD

123 **A Herald Bleed: A Case of Aortoesophageal Fistula and a Review of the Literature**
Graca M. Dores, MD
Marilyn E. Miller, MD
Donald G. Kaufman, MD

128 **Urological Injury and Assessment in Patients with Pelvic Fractures**
David M. Albala, MD
John R. Richardson Jr, MD
John A. Heaney, MD, FRCSI

133 **Diagnosis and Treatment of Bladder Rupture: Characteristics of 42 Cases**
David M. Albala, MD
John R. Richardson, Jr, MD

COLUMNS

103 **EDITOR'S MAILBOX**

138 **HEALTH BY NUMBERS**
Cancer of the Cervix Uteri in Rhode Island

139 **MONTHLY VITAL STATISTICS REPORT**

140 **THE RHODE ISLAND MEDICAL JOURNAL HERITAGE**

143 **UNDERSTANDING CME**

Cover: This month's cover illustration is a painting by Joseph Wilder, MD, entitled "Surgeons at Work". See editorial, page 99.

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AIR FORCE

EDITORIALS

The Art of Joe Wilder, MD

For the first eighty years of its existence, the front cover of the *Journal of the American Medical Association* had been reserved for its table of contents. This utilitarian usage was changed dramatically in 1964 when a color reproduction of James Whistler's mother replaced the customary listing of articles. The editors of the *JAMA*, believing that its reader might not be fully prepared for this precipitous slide into esthetic languor, hastily noted that the painting was chosen as an illustration of a possible iron-deficiency anemia. Since then, however, the *JAMA* covers have unabashedly displayed the great works of Picasso, Rembrandt, Matisse and Van Gogh. But the artist whose works have appeared the most frequently on *JAMA* covers, accounting for eight different covers, is a practicing general surgeon from New York City, Dr Joseph R. Wilder.

Wilder, born in Baltimore some seven decades ago, developed as a youth self-taught skills in lacrosse and attended Dartmouth College on an athletic scholarship. He went on to become Dartmouth's greatest lacrosse player and an All-American as well. Upon graduation he attended Columbia University College of Physicians & Surgeons, completed his residency training at the prestigious New York Hospital, served as a surgeon in the Air Force during World War II and subsequently rose to be chosen chief of surgery at New York's Joint Disease Hospital. Currently, he is professor of surgery emeritus and director of the surgical emergency division at Mount Sinai School of Medicine.

About 30 years ago, at the earnest suggestion of one of his grateful patients, Wilder tried painting as a recreational means to counterbalance the exhausting demands of an intense surgical practice. His initial graphic interests, understandably, were attempts to translate the world of athletics to canvas. His early paintings were of baseball and basketball players, marathon runners, weight lifters and racing cars. In a short time, the closet avocation became a compellingly demanding second career. His days were now divided between predawn painting in his studio and daylight labors in the operating rooms and wards; and in both careers, he prospered.

In the words of the *New York Times*, Wilder is considered by many to be the finest painter of sports images in the world. A collection of his more memorable sports paintings has been assembled in an elegant text, *Athletes*, published in 1985 by H.N.. Abrams, Inc and C.V. Mosby, Co. To quote from its preface, "The strategy, preparations, research, and planning for a painting, an operation, or an athletic contest have so much in common that I am constantly trading off these three worlds, one with the other, favoring none and loving all."

The collected retrospective of Wilder's art in sports is not his sole contribution to the published literature. He is also the author of the widely used *Atlas of General Surgery*.

Wilder's paintings have now been exhibited extensively at numerous art galleries as well as the National Institutes of Health and the National Medical Library in Bethesda.

The cover of this month's *Rhode Island Medical Journal* is an original oil painting by Wilder entitled *Surgeons at Work*, and is one of a number of his large canvases celebrating ". . .the profession of the operating room, the human drama, its emotional content, colors and rhythms. . ."

At times in recent history,

physicians have displayed great collateral skills in expressive writing. The names of physician-authors such as Conan-Doyle, Rabelais, Chekhov and Keats, amongst many others, come to mind. There have been artists, of course, whose works touched frequently on the lives of physicians and their patients, such as Daumier and Kollwitz, but none of the graphic masters had concurrently been practitioners of medicine. Perhaps Wilder's genius will encourage and suggest to his colleagues in medicine that they consider the blank surface of the canvas as a creative outlet. The language of art can indeed provide us with insights, visions and illuminate feelings far beyond mere words. The artist, said Goethe, is like Sunday's child – he alone sees spirits. But, after he has told of their appearing to him, everybody sees them.

Stanley M. Aronson, MD

Should Practicing Physicians Be Routinely Tested For the AIDS Virus?

The impact of AIDS upon the lives of Rhode Island's physicians has yet to be fully realized. The cumulative number of clinically evident cases in the state has now reached 390, but for many practitioners the full import of the disease, and its social ramifications, remains a distant and impersonal prospect.

Behind the seemingly modest numbers of Rhode Island AIDS patients (fewer than 400 recorded cases during the past decade) hide four grave and tangible realities: (1) the disease once begun is uniformly fatal; (2) the disease is communicable; (3) there are many more asymptomatic Rhode Islanders, perhaps as many as 3,000, who are HIV-positive and capable of communicating the virus; and (4) since the AIDS virus is transmitted through biological fluids, physicians and other health workers are at potential risk for infection when working with HIV-positive patients; and as a corollary, the uninfected patient may be at risk if treated by an HIV-positive dental surgeon or physician.

Two recent events bring us closer to the microbiologic and political imperatives of AIDS.

A young patient of an HIV-positive dentist believes that she acquired her AIDS infection during her dental therapy. And a Baltimore surgeon, said to have been infected by one of his patients, has recently died of AIDS.

These two tragedies have prompted the United States Public Health Service to consider a new set of guidelines requiring that physicians and dentists, amongst others, be periodically tested for HIV infection. While it has not as yet been plainly stated, the implication is that those practitioners found to be seropositive will then be forbidden any professional contact with their patients.

Few scientists familiar with the dynamics of AIDS virus transmissibility doubt that a surgeon working with an HIV-positive patient runs a small but measurable risk of incurring infection. The reverse (ie, a surgeon infecting a patient) however, is considered a highly unlikely event; indeed, not a single verified instance of physician-to-patient HIV transmission has been documented.

Given, then, the utterly remote probability that a practicing physician constitutes a possible source of AIDS transmission to the patient, is there any rational public health basis for such blanket testing of physicians? Are not these contemplated guidelines a palpably greater threat to the health of the community by placing impractical and irrational limitations upon the right of physicians to practice their art?

In the words of Gellin and Rogers (vice chair of the National Commission on AIDS), "The message to health professionals will be clear: "To continue to care for your patients, and to use your precious skills which required years of training to obtain, don't take care of the HIV infected or those who you think might be." The negative consequences will far outweigh a doubtful reduction in risk. In a world that already seriously undervalues and trivializes service to others, this would compound the tragedy of AIDS."

In the belief that this question of mandatory testing of physicians is of considerable importance, the

Journal has requested the written opinions of a number of its membership; these responses, as well as unsolicited letters from Rhode Island' s physicians , will appear in one of our next issues.

Stanley M. Aronson, MD

The Providence Medical Journal

Since June, 1989, the *Rhode Island Medical Journal* has been publishing synopses of prior scientific articles and editorials which had appeared in its issues of 25 and 50 years ago. These summary columns in some small measure, reflect the progress of the politics, the science and the humanity of medicine in Rhode Island during the current century. The columns also serve to remind us of how much, and yet how little, we have advanced. On the one hand we may smile indulgently when we read an article written some 50 years ago which, in the light of our current knowledge, seems implausible or naive; and yet in the very next article, we may be humbly impressed with the remarkable insights exhibited by our colleagues half a century ago, despite their lack of the awesome diagnostic and therapeutic facilities which are currently available to us.

The *Rhode Island Medical Journal* began its uninterrupted publication in January of 1917 and hence we cannot provide the reader with the local scientific reasoning of, say 100 years ago. As a partial substitute, however, we shall now summarize the contents of the *Providence Medical Journal*, the legitimate ancestor of the current *Journal*, which had been regularly published between 1900 and 1916. The *Providence Medical Journal* was a much more modest enterprise, appearing but four times a year, and with a yearly subscription price of one dollar. It was edited by George D. Hersey, MD and F.T. Rogers, MD.

Henceforth, the *Rhode Island Medical Journal Heritage* column will summarize items, worthy of current note, which had appeared in issues published 90, 50 and 25 years ago.

Stanley M. Aronson, MD

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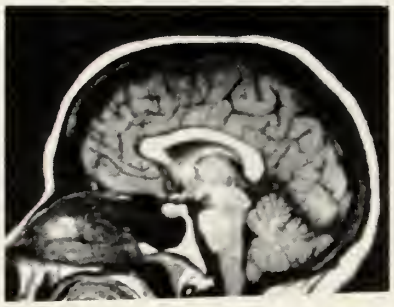

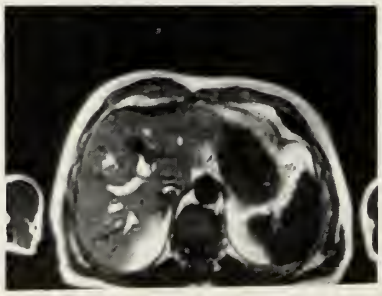

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EDITOR'S MAILBOX

To The Editor:

The following will constitute my reply to Dr Sturim's article on malpractice.

The excellent article by Dr Sturim on malpractice points out the stress inherent in being the subject of a malpractice suit. The Rhode Island Medical Society has instituted a program designed to deal with stress from malpractice suits and other areas in physicians' lives, as well. The program is scheduled to start early spring, 1991, and has been heavily advertised in the *Rhode Island Medical Journal* and the *Rhode Island Medical News*. If there is sufficient demand a second group session can be started. The purpose of these support groups is to enable doctors to deal with the stresses in their lives, including malpractice suits.

In addition, I would like to make one other point. Plaintiffs' attorneys claim that they are doing the public good by getting rid of bad doctors through the process of malpractice suits. I have suggested to attorneys that when they accept a case which seems to show bad practice, that they report this case immediately to the Rhode Island Medical Society or to the Board of Medical Licensure and Discipline for appropriate corrective action. Under the present circumstances in Rhode Island it takes about five years for a case to come to trial. Could an attorney live with his conscience knowing that a doctor might be practicing bad medicine for five years while awaiting trial? I would suggest that the public good is being harmed significantly by the actions of individual plaintiffs' attorneys who do not report in good faith to the Medical Society Peer Review Committees or the Board of Licensure and Discipline the actions of physicians which they think might represent poor practice, so that corrective action can be instituted to protect the public.

Herbert Rakatansky, MD
Impaired Physicians' Committee, Chairman
Past President, Rhode Island Medical Society

ERRATUM :
A statement in the article "*Framework for the Rhode Island Response to the Injury Problem*" that appeared in the December, 1990, vol. 73 *Rhode Island Medical Journal* requires correction. On page 603, within the section on suicide, it is stated that the low suicide rate for children less than 15, as illustrated in Figure 4 is misleading "since suicides are **not** rare for those under 10". In fact, the point to be made here is that suicides for those under 10 **are** rare. Even when self-inflicted death occurs, it is especially difficult to determine intentionality for these youngsters. Therefore, grouping 5-9 year olds with those aged 10-14 understates the suicide rate for the latter group.

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Deciding for Others: Surrogate Decision Making for Incompetent Adults

Dan W. Brock, PhD

The capacities needed for competence in treatment decision-making are three: the capacity for understanding and communication; the capacity for reasoning and deliberation; and the capacity to have and apply a set of values or conception of one's good.

My aim is to sketch an ethical framework for surrogate decision-making about medical treatment for incompetent adults and for thinking about the ethical issues that arise in that decision-making.[1] Surrogate decision-making should seek to extend the ideals of health care decision-making for competent adults, with suitable changes required by and reflecting the patient's incompetence. In the medical ethics literature a substantial, though not universal, consensus has developed that health care decision-making should be shared decision-making between the physician and competent patient. Oversimplifying, the physician brings his or her knowledge, training and experience to the diagnosis of the patient's condition and the prognoses, if different treatment alternatives are followed, including the alternative of no treatment. The patient brings his or her own subjective aims and values to the decision-making process for the evaluation of the alternatives, with their particular mixes of benefits and risks, as spelled out by the physician.[2]

What values underlie a commitment to shared decision-making, and what ends are we seeking to promote with it? I believe there are two central values underlying shared decision-making. The first, and most obvious, is the promotion and protection of the patient's well-being. Shared decision-making rests in part on the presumption that competent patients who have been suitably informed by their physicians about the treatment choice they face are generally, though of course not always, the best judges of what treatment will most promote their overall well-being.[3] The other value is respecting the patient's self-determination. I mean by self-determination the interest of ordinary persons in making significant decisions about their own lives themselves and according to their own values. It is by exercising self-determination that we have significant control over our lives and take responsibility for our lives and the kind of persons we become. These then are the values that guide shared decision-making. For incompetent patients we still want shared decision-making, but then the patient lacks the capacity to participate and a surrogate must take the patient's place. Before considering who should be a surrogate and how the surrogate should decide, the prior question is which patients should have a surrogate to decide for them.

The Determination of Incompetence

In health care this is ultimately a matter of the patient's compe-

Dan W. Brock, PhD, a national authority in the field of biomedical ethics, is Professor of Philosophy at Brown University, Providence, Rhode Island, was staff philosopher to the President's Commission for the Study of Ethical Problems in Medicine, 1981-1982.

ABBREVIATIONS USED:
DPOA: Durable power of attorney

tence to decide for him- or herself. If the patient is competent, the patient is entitled to decide and to give or refuse informed consent to treatment. If incompetent, a surrogate must be selected to decide for the patient. The concept, standards, and determination of competence are complex and there is space here to emphasize only a few important points about competence and incompetence.[4] Adults are presumed to be competent unless and until found to be incompetent. In questionable or borderline cases competence should be understood for two reasons as decision-relative, that is, a patient may be competent to make one decision but not another. The first reason is that different decisions can vary in the demands they make on a patient, for example, in the complexity of the information relevant to the choice. The other reason is that patients can change over time in the decision-making capacities they bring to the decision-making process from the effects on them of medications, their disease, and other factors.

The capacities needed for competence in treatment decision-making are three: the capacity for understanding and communication; the capacity for reasoning and deliberation; and the capacity to have and apply a set of values or conception of one's good. Though these capacities are possessed by people in different degrees, it is important to recognize that the determination of competence is a threshold, not a comparative, determination. This follows from the function that the competence determination plays both in health care and in the law – to sort the patient into either the class of patients who are competent to decide for themselves or into the class of patients who must have a surrogate to decide for them. The crucial question about competence in borderline cases then is how defective or impaired a person's decision making must be to warrant a determination of incompetence. There are two central values at stake in whether a patient is judged competent or incompetent, and so two kinds of errors to be balanced in that determination. One value is protecting the patient from the harmful consequences of his or her choice when the patient's decision-making is seriously impaired. The other value is respecting the patient's interest in deciding for him- or herself when sufficiently able to do so. The two errors to be balanced are failing adequately to protect the patient from the harmful consequences of a seriously impaired choice against failing to permit the patient to decide for him- or herself when the patient is sufficiently able to do so. There is no unique objectively correct balancing of these two values and errors. Instead the proper balancing is inherently an ethically controversial choice.

The evaluation of a patient's competence should address and evaluate the *process* of the patient's decision-making; the standard should not be an outcome standard that simply looks to the content of the patient's choice and whether it is the "correct" choice according to some standard of correctness. Given the values noted above which are at stake for the patient in the competence determination, it follows that the standard for competence should be a variable standard, varying principally according to the consequences for the patient's well-being of accepting his or her choice. The standard should vary along a continuum from high, when the choice appears to be seriously in conflict with the patient's well-being; to moderate, when the patient's choice appears to be comparable to other alternatives in its effect on the patients' well-being; to low, when the choice will clearly best serve the patient's well-being. One controversial consequence of this account of the competence determination is that a patient might be competent to consent to a particular treatment, but not to refuse it, and vice versa. This follows from two facts. First, the process of reasoning to be evaluated will inevitably be different if it leads to a different choice. Second, the effects on one of the values to be balanced–the patient's well-being–if the patient's choice is accepted can be radically different depending on whether the patient has consented to or refused the recommended treatment. Treatment refusal may reasonably trigger an evaluation of the patient's competence, though it should usually trigger as well a re-evaluation by the physician both of the treatment recommendation and of the communication of that recommendation to the patient.[5] The basic question the competence evaluator should be trying to answer is: does the patient's choice sufficiently accord with the patient's own underlying and enduring aims and values for it to be accepted and honored, even if others, including the physician, may think it not the best choice and not the choice that they would make?

The Selection of a Surrogate

Let us now assume that a patient has been found to be incompetent to make a particular treatment choice so that a sur-

rogate must act for the patient. There are now two central questions: who should serve as surrogate for the patient? What standards should guide the surrogate's decision? I will first take up selection of a surrogate. If we are to respect the incompetent patient's wishes as best we can, then the surrogate should be the person that the patient would have wanted to act as surrogate for him or her . In a number of states, it is possible for a person, while competent, to designate legally a surrogate who will make health care decisions for him or her in the case of later incompetence by executing a Durable Power of Attorney for Health Care (DPOA). This document also allows, but does not require, a person to give instructions to the surrogate about one's wishes concerning treatment. Ethically, even if not legally, all oral designation by a person of a surrogate should have nearly the same weight as a formal DPOA. Patients should be encouraged while competent to designate who will decide for them in the case of later incompetence, and physicians have a responsibility to seek out this information from their patients early in their patients' treatment and while patients are still competent, especially if a period of later incompetence is relatively predictable.

The Durable Power of Attorney . . . allows, but does not require, a person to give instructions to the surrogate about one's wishes concerning treatment.

In most cases at present, an incompetent patient will not have explicitly designated who is to serve as his or her surrogate. It is then reasonable to act on a presumption that a close family member of the patient, when available, is the appropriate surrogate.[6] This is also the common practice in health care. There is space here to mention only the most important considerations that support this presumption for a family member acting as surrogate. First, usually a family member will be the person the patient would have wanted to act as surrogate for him or her. Second, usually a family member will be able to arrive at the best decision for the patient because the family member will know the patient best and will be most concerned for the patient's welfare. It is important to underline that the claim is only that the practice of using a close family member as surrogate will result overall in better decisions for patients than any feasible alternative practice, such as appointing an attorney to act as the patients' guardian. Third, the family is a central social and moral unit in our society to which important responsibilities are assigned and within which important values are realized. The family has important responsibilities to care for its dependent members. While dependent children are the most obvious example, dependent adults are another important instance of this responsibility. The family is also the main place in which most persons in our society are able to pursue and realize the values of intimacy and privacy. Both to fulfill these responsibilities and to realize these values the family must be accorded significant, though not unlimited, freedom from external oversight, intrusion, and control.

It is to be emphasized that these and other grounds for the presumption for a close family member to be an incompetent patients' surrogate do not require that the surrogate always make the best or optimal choice. That would be too high and unrealistic a standard. On the other hand these grounds for the family as surrogate should also make clear that family members' authority as surrogate decision makers is not unlimited. The presumption for family members as surrogates can be rebutted in particular cases in which these grounds do not hold–for example, there is no close relation between patient and family member, or there is a clear conflict of interest between patient and family member. In such cases, an incompetent patient's physician can have a positive responsibility not to allow the family member to act as surrogate, which can include appeal to the courts to have another appointed as surrogate if that proves necessary.

. . . the family is a central social and moral unit in our society to which important responsibilities are assigned and within which important values are realized.

Sometimes there is no family member available to serve as surrogate. When a close friend is available and willing to serve as surrogate, then most of the reasons that support a family member as surrogate will support using the friend, though if especially controversial or problematic decisions must be made physicians may want to have the friend be formally appointed as guardian by the courts. In other cases there is no family member or friend available to serve as surrogate. Health care institutions vary as to how

they handle such cases and flexibility both within and between institutions is desirable because of the wide range of different decisions that must be made. What is important is that institutions have a settled and public policy for such cases so that decision-making does not become paralyzed from lack of a natural surrogate. A hospital might have, for example, a policy that for a specified range of decisions to forego life-sustaining treatment or resuscitation, the attending physician's proposed decision would be referred to the Chief of Service who could review the decision and take any further steps he or she deemed appropriate, such as referral to an ethics committee or to the courts. At least for decisions about life-sustaining treatment, some involvement of one or more others besides the attending physician is desirable when the patient lacks a family member or friend to act as surrogate.

Guidance Principles for Surrogate Decisions

What standards should a surrogate employ in deciding for an incompetent patient? While it is oversimplified in some respects that I will address below, one can think of three ordered guidance principles for surrogates to employ in decision-making: Advance Directives, Substituted Judgment, Best Interests. These are ordered principles in the sense that the surrogate should employ the first if possible, or if not it, then the second if it is possible, and if neither the first or second can be used, then the third.

The Advance Directives principle tells the surrogate that if a valid advance directive exists that the patient gave while competent specifying how the decision at hand should be made, there is a strong presumption that it should be followed. Formal advance directives take two principal forms, so-called Living Wills and DPOAs. Living Wills are given legal force in approximately 40 states, while DPOAs have legal force in far fewer jurisdictions at present.

At least for decisions about life-sustaining treatment, some involvement of one or more others beside the attending physician is desirable when the patient lacks a family member or friend to act as surrogate.

Living Wills have several common features that limit their usefulness, of which I shall mention two. First, they are nearly always formulated in vague terms both as to the patient conditions in which they apply,, such as "terminally ill," and as to the treatments they cover, such as "extraordinary measures" or "aggressive treatment". This is to some extent inevitable when they are executed well in advance of the decision to be made and means that there is commonly significant interpretation that others must undertake as to how they should now apply. Second, and probably in response to worries about potential abuses, enabling statutes in some states place two kinds of limitations on living wills; in which they can be executed, for example, the person must already have been diagnosed as terminally ill with death imminent; limitations in the circumstances in which they apply, for example, the decision cannot cover nutrition and hydration. When persons follow these restrictions and limitations in the hope of giving living wills legal enforceability, there will be many circumstances in which they fail to apply. Since Living Wills are relatively rarely brought into court for enforcement, but more often function as a means of informing others about the patient's wishes, restrictive formulations may be inadvisable.

DPOAs are the more desirable form of advance directive for most persons. First, they commonly allow a person to give more detailed instructions to the surrogate about his or her wishes regarding treatment, though it is important to avoid letting greater detail narrow the application of the instructions. Second, they respond to the importance of the task of later interpreting the patient's instructions by allowing the patient to designate who will do that for him or her. DPOAs will not be the preferable form of advance directive, of course, for persons who have no one to name to act as surrogate for them.

I noted above that there is a strong presumption that a valid advance directive should be followed, but did not claim it should be absolutely binding. This is because there are reasons why advance directives should not have ethically the same degree of binding force as the contemporaneous decision of a competent patient. In the case of a competent patient, the decision can be made attending to the full and detailed context, whereas advance directives must inevitably be formulated before the precise context of future decisions is known. This means that occasionally a treatment decision will arise in circumstances radically different from those assumed likely by

the patient when executing the advance directive; in such cases, following the letter of the directive may be contrary to following its spirit or the intentions of the patient. Moreover, the worry about advance directives that the patient might have changed his or her mind about treatment when the time came actually to decide does not arise for a competent patient able to decide. Finally, a decision by a competent patient that appears significantly contrary to the interests of that patient commonly will be opposed by others who care for the patient, thereby testing the understanding and resolve of the patient to an extent not possible with advance directives. Despite these limitations, there are good reasons in practice to accord a strong presumption to any patient's advance directive about care.

At the present time, and probably for the foreseeable future, most patients do not have advance directives and then the Substituted Judgment guidance principle should be followed. This principle tells the surrogate to attempt to decide as the patient would have decided, if competent, in the circumstances that now obtain. In effect, the principle directs the surrogate to use his or her knowledge of the patient and of the patient's aims and values to infer what the patient's choice would have been.

Physicians have an important responsibility in helping surrogates to understand their role in applying substituted judgment. The physician's approach should not be of the form, "What do you now want us to do for your mother?" but instead, along the following lines: "you of course knew your mother better than I did, so help us decide together what she would have wanted done for her now." This substituted judgment approach helps get decisions more in accordance with the patient's wishes, thereby better fulfilling the values underlying shared decision-making.[7] It also has the practical advantage of leaving the responsibility for the choice made as much as possible with the patient, thereby often making the psychological and emotional burdens of surrogates easier to carry and facilitating their effective participation in decision-making. The answer when life-sustaining treatment is in question of "Do everything" is nearly always unhelpful and to be avoided. Surrogates should always be assured that all appropriate care will always be given, including all care needed to maintain the patient's comfort and dignity. But what care is appropriate will often change with changes in the patient's condition and prognosis, and surrogates must be helped to understand that all possible care is not automatically appropriate care.

The principle (of Substituted Judgment) directs the surrogate use his or her knowledge of the patient and of the patient's aims and values to infer what the patient's choice would have been.

Two features of substituted judgment decision-making should be explicitly noted. First, it will let surrogates take account of how the interests of others besides the patient will be affected by the decision to be made, though only to the extent that there is evidence the patient would have given that weight to those interests. Second, it will let surrogates making decisions weigh considerations about the quality of life of the patient, both at present and as it will be affected by treatment decisions, though only quality of life for that patient and according to the patient's own values. When foregoing life-sustaining treatment is in question only a very limited quality of life judgment is relevant, viz. is the best quality of life possible for the patient with life-sustaining treatment sufficiently poor that the patient would have judged it to be worse than no further life at all? No judgments about social worth or the social value of the patient are warranted under substituted judgment.

When there is no information available about what this particular patient would have wanted in the decision at hand, the Best Interests principle directs the surrogate to select the alternative that best furthers the patient's interests. This in effect amounts to asking how most reasonable persons would decide in these circumstances, which is justified by the absence of any information about how this patient differs from others. Treatment choices based on best interests can be especially difficult in the common circumstances in which choice is from a range of alternatives about which reasonable persons can and do disagree. Because of this difficulty, it is important where possible to avoid having to appeal to best interests by engaging in advance planning with patients when they are still competent to determine their wishes about future treatment options.

I noted above that a strict ordering of these guidance principles was an oversimplification and we are in a position now to

see one reason why that is so. Evidence bearing on the patient's wishes concerning the decision at hand, whether from an advance directive or from the surrogate's knowledge of the patient, is neither fully determinate and decisive on the one hand, or completely absent on the other. Instead, such evidence ranges along a broad continuum in how strongly it supports a particular choice. In all cases, physicians and surrogates should seek confidence that the choice made is reasonably in accord with the patient's wishes or interests. The better the evidence about what this particular patient would have wanted, the more one can rely on it. The less the information and evidence about what this patient would have wanted, the more others must reason in terms of what most persons would want.

What constitutes adequate evidence for surrogates to have about the patient's wishes has been an issue in several recent court cases, most notably in the recent O'Connor case in New York and in the Cruzan case in Missouri, which was recently upheld by the US Supreme Court.[8] In the New York case, where the standard was spelled out more, the court imposed an extremely high standard of evidence for foregoing life-sustaining treatment by a surrogate, requiring clear and convincing proof that the patient had made a settled commitment while competent to reject the particular form of treatment under circumstances such as those now obtaining. Where there is any significant doubt, the court reasoned, the decision must be on the side of preserving life. This decision set a very difficult standard in New York for patients and their surrogates to satisfy, and thereby establishes a very strong presumption in favor of extending the lives of incompetent patients with life-sustaining treatment. The US Supreme Court in Cruzan upheld the right of the state of Missouri to impose this same very strong presumption, though without endorsing the wisdom of doing so.

Such a presumption underweights patients' interest in self-determination, and fails as well to recognize adequately the extent to which patients' well-being is determined by patients' own aims and values. But isn't it reasonable, the Court might ask, always to err on he side of preserving life when there is any doubt about the patient's wishes? At a time several decades ago when medicine only rarely had the capacity to extend life in circumstances where doing so would have been unwanted by, and not a benefit to, patients, such a policy would have been reasonable. It is a commonplace, however, that medicine has gained vastly enlarged capacities to extend life in recent decades. Patients' lives can now often be extended when they would not want this done, and as a result New York's and Missouri's very strong presumption in favor of extending life when there is any significant doubt about the patient's wishes can no longer be justified. Though the US Supreme Court found no constitutional bar to the clear and convincing evidence standard, there remain good reasons for other states not to adopt it.

Conclusion

Having briefly sketched an ethical framework for surrogate decision-making for incompetent patients, I want to conclude with a plea for "preventive ethics" aimed at reducing as much as possible the necessity to resort to surrogate decision-making when the patient's wishes are unknown or significantly uncertain. This can only be done by persons, while still competent, talking with their physicians and families about their treatment wishes should the become seriously ill. This is especially appropriate in the case of persons with chronic, progressive diseases in which both a possible period of incompetence and the nature of later treatment decision likely to arise are relatively predictable. Physicians have an important role in making this prior planning occur substantially more often than it now does by encouraging their patients to reflect about their wishes and to make those wishes known to others likely to be involved in treatment decisions for them. The need for surrogate decision-making in health care will never be eliminated, nor can all difficult decisions be avoided, but it should be possible greatly to reduce the number of cases in which physicians and surrogates must decide about an incompetent patient's care lacking knowledge about the patient's wishes that could have been obtained earlier, had it been sought.

References

[1] I draw freely here on prior published work I have done on this topic, some of it collaborative work with Allen Buchanan. See especially Allen E. Buchanan and Dan W. Brock, Deciding for Others: The Ethics of Surrogate Decision-Making (Cambridge: Cambridge University Press, 1989).

[2] Some respects in which this division of labor is oversimplified are explored in my paper, "Facts and Values in the Physician/Patient Relationship," in Ethics, Trust and the Professions, eds. R. Veatch, T. Langen, E. Pellegrino (Washington, DC: Georgetown University Press, 1991).

[3] For a discussion of different kinds of cases in which competent patients make irrational choices, and the responsibilities of their physicians in such circumstances,

see Dan W. Brock and Steven A. Wartmen, "When Competent Patients Make Irrational Choices," *New England Journal of Medicine*, 322: (May 31) 1990, 1595-1599.

[4] See especially, Buchanan and Brock, *op. cit.*, Ch. 1.

[5] Some studies have shown that the most common cause of treatment refusals is failure of the communication process between physician and patient, and that most refusals are consequently withdrawn when the recommendation is better explained so that the patient understands it adequately. Cf. PS Applebaum and LS Roth, "Treatment Refusal in the Medical Hospital," in *President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research, Making Health Care Decisions: The Ethical and Legal Implications of Informed Consent in the Patient-Practitioner Relationship: Vol. 2 Appendices.* (Washington DC: US Government Printing Office, 1982).

[6] In most jurisdictions, a close family member lacks explicit legal authority to act as the patient's surrogate until formally appointed by a court as the patient's guardian. States should consider adopting legislation like the recently enacted Health Care Decisions Act in the District of Columbia which formally authorizes a family member to act as surrogate for an incompetent patient without recourse to guardianship proceedings.

[7] Confirmation that asking the right question matters in the choices surrogates make can be found in Tom Tomlinson, et. al., "An Empirical Study of Proxy Consent for Elderly Persons," *The Gerontologist 30*, 1 (1990) 54-60.

[8] In re O'Connor, No. 312 (N Y Court of Appeals, October 14, 1988, Cruzan v. Director, Missouri Dept. of Health, 110 S. Ct. 2841, 1990).

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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Acquired Immune Deficiency Syndrome and the Surgeon

Steven Schechter, MD
Joseph Amaral, MD

By and large, the treatment of abdominal pain in AIDS patients is nonsurgical.

Considerable attention has been placed on the role of the surgeon and the surgical team in the management of surgical problems in HIV+ and AIDS patients. These reports have focused on the risks and prevention of HIV infection in the members of the surgical team when managing HIV+/AIDS patients. In contrast, less attention has been paid to the unique surgical problems HIV+/AIDS patients present to the surgeon. In addition to the garden variety pathology seen in non-HIV+/AIDS patients in similar age groups, HIV+/AIDS patients manifest unusual opportunistic infections and rare malignancies.[1] Because of their immuno-compromised state, the signs and symptoms of peritonitis from an abdominal catastrophe may be masked. The following is an analysis of the role of the general surgeon when confronted with an HIV+/AIDS patient with abdominal pain.

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By and large, the treatment of abdominal pain in AIDS patients is nonsurgical. Several large studies from urban high-risk areas support this statement[2-4] LaRaja et al studied 904 AIDS admissions at the Cabrini Medical Center in New York City over a three year period and found only 36 patients requiring abdominal exploration (4%). The percentage of AIDS patients requiring celiotomy at the Kaiser Permanente Medical Center was similar.

Gastrointestinal infections probably account for at least half of all complaints of abdominal pain. Infections caused by organisms such as Cryptosporidium, Giardia lamblia, Amebiasis, Salmonella, and Campylobacter jejuni are common. Thus, stool specimens for culture and microscopic examination are essential.

Ileus and hepatosplenomegaly, which are both common in AIDS patients, may mimic an acute abdomen. Barone, et al from New York City found that 70% of AIDS admissions with abdominal pain had nonsurgical diseases. Nevertheless, an aggressive diagnostic approach is warranted in the immuno-compromised host with subtle complaints or findings on abdominal examination. Computer assisted tomography, ultrasound, and nuclear scans are valuable non-invasive techniques. However, the presence of polymicrobial sepsis may be the only clue to an intra-abdominal problem.

The etiology of the acute abdomen in HIV+ and AIDS patients may be broadly divided into two categories: common disorders and those associated with the immuno-compromised state.[5] The old adage that uncommon manifestations of common diseases occur more commonly than uncommon diseases remains true. Appendicitis, cholecystitis, or diverticulitis should not be overlooked. Potter et al

ABBREVIATIONS USED:
AIDS: acquired immune deficiency syndrome.
CMV: cytomegalovirus
HIV+: positive for human immmunodeficiency virus
IgG: immune globulin G
ITP: immune thrombocytopenic purpura

described 5 patients with AIDS who required surgical evaluation for abdominal pain in which an unusual spectrum of infectious and neoplastic intra-abdominal pathology was present.

Foremost among infectious agents causing abdominal complaints in the HIV+/AIDS patient is cytomegalovirus (CMV). Cytomegalovirus infection is common in the immuno-compromised host and may be histologically evident in many organ systems. Although the clinical significance may not always be clear, there is associated morbidity and mortality. Peritonitis secondary to CMV enterocolitis and perforation is well described. Cytomegalovirus is thought to infect gut endothelial cells leading to vasculitis and subsequent perforation. The importance of CMV as a pathogen in AIDS patients is seen in the work of Wilson et al. These investigators reported 14 emergent celiotomies on AIDS patients of which 11 were due to perforations. CMV infection was incited in 8 of these perforations.[6]

. . . uncommon manifestations of common diseases occur more commonly than uncommon diseases. . .

Another group of unusual infections seen commonly in HIV+/AIDS patients are those caused by the Mycobacteria. More than half of the abdominal mycobacterial infections at Bellevue Hospital were found in AIDS patients.[8] Mycobacterium avium intracellulare infections can manifest as hepatosplenomegaly, retroperitoneal adenopathy, or as an intra-abdominal inflammatory mass. These mycobacterial organisms may lead to active disease in AIDS patients as a result of a primary infection or as a result of reactivation when cellular immunity is depressed. A trial of antibiotics may be warranted in thi ssetting prior to any operative intervention.

Infections with unusual organisms in the HIV+/AIDS patient are not limited to the enteric viscera. AIDS patients, especially in the intensive care unit setting, have been reported to develop acalculous cholecystitis. Although several reports have identified CMV within the gallbladder mucosa, the significance remains to be determined. Cryptosporidium has been identified as a possible etiologic agent.[11] Overall, the mortality of acalculous cholecystitis in the AIDS patient is quite high. The Cabrini Medical Center experience with acalculous cholecystitis revealed a mortality of 33%. In part, this is the result of delays in diagnosis and therapy. However, equally or more important is the high mortality associated with emergency operations in AIDS patients. For this reason, acute acalculous cholecystitis may be best treated by percutaneous drainage of the gallbladder rather than by an open operative procedure that will require general anesthesia.

Foremost among the malignancies noted in HIV+/AIDS patients is the development of non-Hodgkin's lymphoma. These may present in the gastrointestinal tract with obstruction, bleeding, or perforation (the latter usually following chemotherapy or radiotherapy). Another common malignancy in HIV+/AIDS patients is Kaposi's sarcoma which can be seen endoscopically in 40% of AIDS patients.[7] This neoplasm of the reticuloendothelial system is much more aggressive when associated with AIDS. Although these lesions may obstruct, bleed, or serve as an intussusception point, the majority are clinically asymptomatic.

Immune thrombocytopenic purpura (ITP), an immune-mediated disease whereby platelets are destroyed, has an increased incidence in AIDS patients. Although the exact mechanism for classic ITP has not been clearly identified, circulating IgG antibodies against platelet antigens probably play a major role with subsequent splenic sequestration and destruction. Spleens are rarely enlarged although in HIV related ITP, splenomegaly is more common. As is the case for classic ITP, failure to respond to steroids or inability to withdraw steroids is an indication for splenectomy. Schneider et al reported a 60% response rate to splenectomy with a mean follow-up of one year in a group of 15 HIV+ homosexual males and others have reported response rates in excess of 90%[10]. Furthermore, evidence suggests removal of the spleen in HIV+ patients does not lead to more rapid development of AIDS in these patients. In this regard, it is noteworthy that splenectomy in HIV+ patients with AIDS is less likely to be beneficial and is thought to be contraindicated because of the high surgical risk and limited prognosis.

. . . wound healing and resistance to wound infection appear to be normal or at least not significantly altered in HIV+ patients who have not developed AIDS.

Since the two major risk groups for acquiring HIV infections are homosexuals and intravenous drug users, it is not

surprising that surgical disorders common in these groups of patients will also be common in HIV+/AIDS patients. For example, arterial aneurysms, deep venous thrombosis and septic thrombophlebitis are common in HIV+ and AIDS patients. Similarly, anorectal disorders including condylomata, perirectal abscesses, fistula in ano and hemorrhoids are very common. In general, all these disorders are responsive to the usual therapies. Caveats in the management of these disorders include: careful elimination of the laser or electrocautery plumme during treatment of condyloma because of the risk of airborne contamination with the virus; avoidance of banding of hemorrhoids in AIDS patients because of the reported development of necrotizing infections following banding; avoidance of large incisions in the perirectal area of AIDS patients since wound healing in this area is often impaired. Anal warts and abscesses have been associated with neoplasia with young homosexuals.[12]

Based on limited data, wound-healing and resistance to wound infection appear to be normal or at least not significantly altered in HIV+ patients who have not developed AIDS.[13] In contrast, both these functions appear to be significantly impaired in the patient with AIDS.

Wilson et al found elective and emergent abdominal surgery in AIDS patients to carry a 9% and 46% mortality respectively.

Ultimately, the goal in the surgical management of the AIDS patients is to intervene prior to the development of an emergency. Wilson et al, found elective and emergent abdominal surgery in AIDS patients to carry a 9% and 46% mortality respectively. Indeed, emergent celiotomy has been associated with a mortality rate as high as 70% in AIDS patients. Death in the majority of these patients is the result of the complications of AIDS infection itself, with most deaths resulting from *Pneumocystis carinii* pneumonia and respiratory failure.

References

[1] Gottlieb MS, Schroff R, Schanker HM, et al. *Pneumocystic carinnii* pneumoni and mucosal candidiasis in previously healthy homosexual men: evidence of a newly acquired cellular immunodeficiency. N Engl J Med. 1981; 305: 1425-1431

[2] Barone JE, et al. Abdominal pain in patients with acquired immune deficiency syndrome. Ann Surg 1986 Vol. 204, No 6: 619-623.

[3] Nugent P, O'Connell TX. The surgeon's role in treating acquired immunodeficiency syndrome. Arch Surg. Vol. 121 Oct 1986 1117-1120

[4] LaRaja RD et al. The incidence of intra-abdominal surgery in acquired immunodeficiency syndrome: a statistical review of 904 patients. Surgery 1989; 105: 175-9.

[5] Nylander Jr WA. The acute abdomen in the immuno-compromised host The surgical clinics of North America. Vol. 68 No 2 April 1988 page 457-467

[6] Wilson SE et al. Acquired immune deficiency syndrome (AIDS) Ann Surg 1989, Vol 210 No 4 428-433

[7] Friedman SL, Wright TL, Altman DF. Gastrointestinal Kaposi's sarcoma in patients with AIDS. Endoscopic and autopsy findings. Gastroenterology 1985; 89: 102-108

[8] Rosengart TK, Coppa "GF. Abdominal mycobacterial infections in immunocompromised patients. The American Journal of Surgery Jan 1990; 159: 125-131

[9] Burack JH, Mandel MS, Bizer LS. Emergency abdominal operations in the patient with acquired immunodeficiency syndrome. Arch Surg March 1989; 124: 285-286.

[10] Schneider PA, Abrams DI, Rayner AA, Hohn DC, Immunodeficiency-associated thrombocytopenic purpura (IDTP). Arch Surg 1987; 122: 1175-1178.

[11] Kavin H, Jonas RB, Chowdhury L, Kabins S. Acalculous cholecystitis infection in the acquired immunodeficiency syndrome. Annals of Internal Medicine. January 1986; 104: 53-54.

[12] Miles, Hag et al. Surgical management of anal-rectal disease in HIV-positive homosexuals. BrJsurg 1990; 77:869-71.

[13] Buehrer, JL et al. Wound infection rates after invasive procedures in HIV-1 seronegative hemophiliacs. AnnSurg 1990, 211: 4, 492-498.

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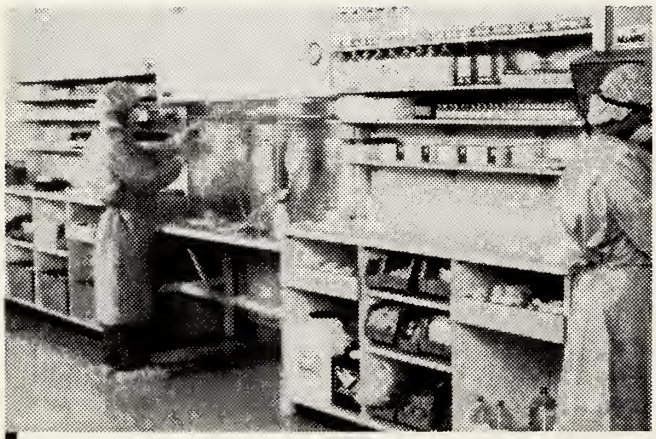

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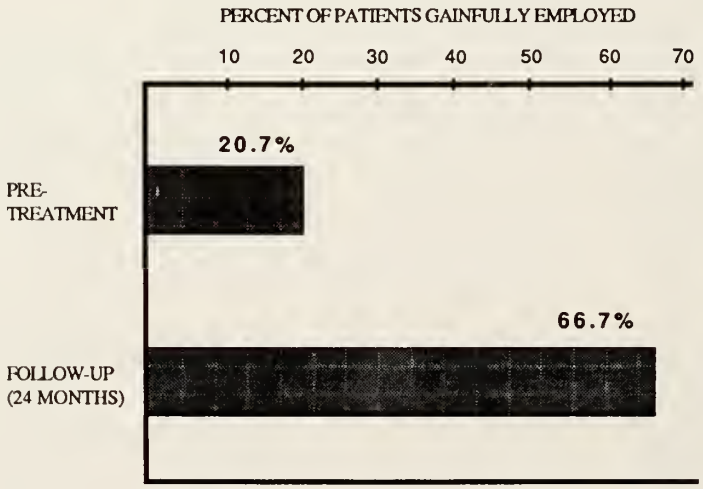

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The Clinical Use of Digibind in Digitalis Toxicity

Richard V. Morgera, MD

Digibind is antigen-binding fragments (Fab) derived from specific antidigoxin antibodies raised in sheep.

Digoxin has a relatively narrow therapeutic range (0.5 - 2.0 ng/ml) making patients susceptible to life-threatening toxic complications from therapeutic, accidental or deliberate ingestion. In June 1989, Digibind (Burroughs Wellcome Co) was approved by the FDA as the first biological product developed and released as a specific drug antidote. Digibind is antigen-binding fragments (Fab) derived from specific antidigoxin antibodies raised in sheep. These antidigoxin antibodies are derived by immunizing sheep with a conjugate of digoxin and human albumin. The antidigoxin antibodies are then cleaved by papain to form an Fc fragment and two monovalent Fab fragments which are then isolated.[1]

Digibind will bind molecules of free digoxin in the extracellular fluid making them unavailable at their subcellular site of action, the Na-K-ATPase receptor. The resultant decrease in extracellular digoxin creates a concentration gradient causing the further egress of intracellular and Na-K-ATPase-bound digoxin into the extracellular space where it is readily bound by Digibind. The affinity of Digibind for digoxin is one hundred times greater than digoxin's affinity for the Na-K-ATPase receptor. For digitoxin, the affinity is ten times greater for Digibind than the same receptor.[2] The bound Fab-digoxin complex is then excreted by the kidney. Complete elimination of the complex may take a week or longer if there is renal impairment.

The current indication for the use of Digibind includes the treatment of life-threatening digoxin intoxication but does not include milder cases of digitalis toxicity or the asymptomatic patient with elevated serum digoxin levels. Use in potentially life-threatening cases must be guided by clinical judgment. The following are acceptable guidelines for Digibind use.[3]

1) Digitalis-induced arrhythmias, including ventricular tachycardia, ventricular fibrillation and high-degree AV block.
2) The single ingestion of greater than 10 mg of digoxin in a healthy adult.
3) The single ingestion of greater than 4 mg of digoxin in a healthy child.
4) Digitalis intoxication with a potassium level greater than 5 meq/L.

The use of Digibind should follow or closely parallel the institution of standard treatment guidelines for digitalis intoxication. These include gastric lavage and charcoal administration, when acute digoxin ingestion presents early, and anti-arrhythmic drugs including Phenytoin and Lidocaine. When Digibind is administered, studies have shown an anti-arrhythmic response in fifteen to thirty minutes. If there is no improvement within this time frame, then

ABBREVIATIONS USED:
AV: atrioventricular
Fab: antigen binding fragments
FDA: Food and Drug Administration
meq/L: milliequivalents per liter
Na-K-ATPase: Sodium-potassium-adenosine triphosphatase
TBL: total body level

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the diagnosis of digoxin toxicity should be reconsidered or additional Digibind administered.

It is of note that Ipecac may be relatively contraindicated in acute digoxin overdose because of its propensity to increase vagal tone thus acting synergistically with digoxin's AV nodal-blocking affect to produce profound bradycardia.[4]

Since massive acute doses of digoxin will poison the membrane-bound Na-K-ATPase system, hyperkalemia may result from potassium's shift to the extracellular location. The acute treatment of hyperkalemia in digoxin toxicity should include the rapid administration of intravenous sodium bicarbonate, as well as insulin and dextrose to facilitate potassium reentry into the cell. Potassium levels will drop within one to five hours and need to be closely monitored for the development of hypokalemia.

The use of Digibind should follow or closely parallel the institution of standard treatment guidelines for digitalis intoxication.

Digibind is administered via intravenous infusion over thirty minutes through a 022 micron filter. The dosage is calculated by estimating the total body load of digoxin and remembering that one vial of Digibind (40 mg) will bind 0.6 mg of digoxin or digitoxin.[6,7] The average dose in clinical trials has been 40 mg or 10 vials. The dosage ingested and the appropriate Digibind are included in each Digibind package insert. The three steps for calculation of needed Digibind dosage are listed below.

CALCULATIONS TO DETERMINE DIGIBIND DOSAGE

1. CALCULATIONS OF TOTAL BODY LEVEL (TBL):

   a. Calculation of TBL from a known ingested dose.
   Digoxin ingested (mg)x 0.8 (% bioavailability) = TBL digoxin (mg)
   Digitoxin ingested (mg)x 1.0 (% bioavailability) = TBL digitoxin (mg)

   b. Calculation of TBL from a known serum digoxin concentration (SDC). (Remember that a steady-state SDC does not occur until 6 to 8 hours after the last digoxin dose. Also, remember that the estimated volume of distribution for digoxin is 5.6 liters and for digitoxin, 56 liters.)

$$\frac{\text{SDC x estimated volume of distribution x patient's weight (kg)} = \text{TBL (mg)}}{1000}$$

2. CALCULATION OF THE NUMBER OF DIGIBIND VIALS TO BE ADMINISTERED:

$$\text{Digibind dose (vials)} = \frac{\text{TBL (mg)}}{\text{0.6 mg digoxin/vial of Digibind}}$$

3. If after acute ingestion, neither the amount ingested nor an accurate serumdigoxin concentration is known, 20 vials of Digibind is a proper dose.[7] This amount will adequately treat most life-threatening symptoms of digitalis toxicity.

Digibind administration is associated with a very low incidence of allergic manifestations. With this in mind, caution should be exercised in patients with a known allergy to sheep products or who have been previously treated. The major adverse effects of Digibind administration result from the sudden withdrawal of digitalis actions including loss of both its chronotropic and inotropic effects. This may result in the return of a rapid ventricular response in patients with atrial fibrillation or the worsening of congestive heart failure in patients with underlying cardiac decompensation. Profound hypokalemia may result after Digibind treatment and serum levels must be closely monitored. The serum digoxin concentration will increase after administration of Digibind but this level represents the inactive Fab/digoxin complex. Thus, routine digoxin levels are not helpful during this period.

Thirteen acute care hospitals in Rhode Island were surveyed to determine the availability of Digibind on an emergent basis. Five hospitals did not stock Digibind at all.[7] Some hospitals felt that they could borrow the product from another hospital if it was needed. The replies indicated that the time frame that Digibind would need to be administered on an emergent basis was not appreciated nor was the amount needed for treatment of acute intoxication appreciated.

This medication should be available in adequate amounts in all acute care Rhode Island Hospitals.

One hospital pharmacy director cited cost as the reason for stocking only one vial of

Digibind. Attention must be paid towards Burroughs Wellcome's returnable good policy in which the company will replace, free of charge, unused Digibind vials up to one year past the expiration date. Thus, an initial purchase of ten vials will be sufficient for most emergent needs, but if unused, Digibind can be replaced and updated at no additional cost.

Summary

It is clear that Digibind needs to be administered on an emergent basis for acute digoxin ingestion and digoxin-induced lethal arrhythmias with or without hyperkalemia. This medication should be available in adequate amounts in all acute care Rhode Island hospitals.

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References

[1] Clarke, W., Acute Digoxin Overdose: Use of Digoxin-Specific Antibody Fragments. American Journal of Emergency Medicine 1988; Volume 6, Number 5; pp. 465-469.
[2] Wenger, T., The Data on Digibind. Emergency Medicine June 15, 1987; pp. 109-111.
[3] Nugent P, O'Connell TX. Clarke, W., Acute Digoxin Overdose: Use of Digoxin-Specific Antibody Fragments. American Journal of Emergency Medicine 1988; Volume 6, Number 5; pp. 465-469
[4] Kulgi, K., Countering Digoxin Toxicity. emergency Medicine June 15, 1987; pp. 114-118.
[5] Smith, T.,, Recognition and Management of Digitalis Intoxication Symposium. Montreal, Canada July 23, 1988.
[6] Martiny, S., Treatment of Severe Digitalis Intoxication with Digoxin-Specific Antibody Fragments: A Clinical Review 1988; Volume 16, Number 6; pp. 629-634.
[7] Digibind Product Information, Burroughs Wellcome Company, Research Triangle Park, NC 1988.

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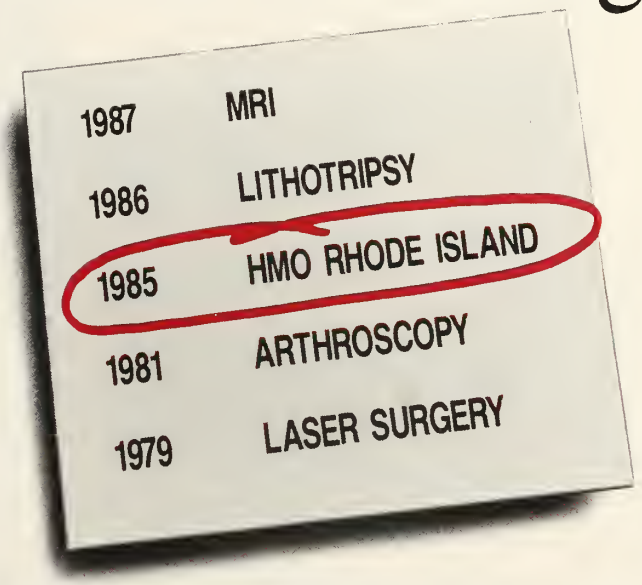

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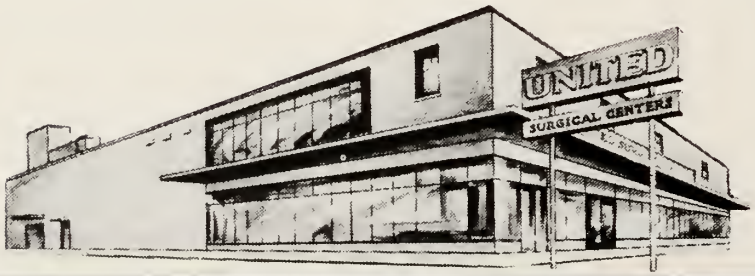

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A Herald Bleed: A Case of Aortoesophageal Fistula and A Review of the Literature

Graca M. Dores, MD
Marilyn E. Miller, MD
Donald G. Kaufman, MD

The majority of aorto-esophageal fistulas are caused by thoracic aneurysms.

Aortoesophageal fistula is a rare cause of upper gastrointestinal bleeding, and despite its consistent presentation of midthoracic pain, "sentinel" hemorrhage, and final exsanguination, the diagnosis is rarely made before autopsy. Awareness of the entity and early surgical intervention continue to be the most important factors in successful management of aortoesophageal fistulas. We present a case and review the literature on this rare clinical entity.

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Case Report

A 78-year-old, white male with an unremarkable past medical history was admitted to the hospital after being found on the floor in a "pool of blood". The patient had no complaints other than feeling weak after falling at home. He admitted to smoking six cigars a day, but denied alcohol consumption or taking any medications. A relative, however, noted that the patient had recently been taking aspirin-like products "by the handful" for "stomach pains".

In the emergency room the patient's blood pressure was 120/70 and pulse 88 per minute, without orthostatic changes. Significant findings included a thin, deaf, white male in no acute distress. His abdomen was soft, and brown stool was positive for occult blood. An estimated two liters of bright, red blood were initially removed with nasogastric lavage. The hemoglobin was 10.6 g/dl, and there was no evidence of coagulopathy. Gastroscopy was performed but did not identify an active bleeding site; much of the view was obscured by a tremendous amount of clot in "the stomach". The patient was treated with blood products, hydration, H2-blockers, and antacids. The hemoglobin slowly continued to decrease.

Endoscopy performed on the third hospital day demonstrated an erosive gastritis of the antrum and prepyloric area of the stomach. The patient remained stable until the sixth day when he had recurrent upper gastrointestinal (GI) bleeding associated with profound hypotension. Endoscopy was attempted, but the view was again obscured by a "tremendous amount of clot in the stomach". Blood was noted in the esophagus, but the gastroesophageal junction and lower-mid esophagus showed no abnormality. An exploratory laparatomy was

ABBREVIATIONS USED;
AEF: aortoesophageal fistula
GI: gastrointestinal

undertaken to identify the site of bleeding. The stomach was noted to be "massively distended with blood clot" and had multiple, small, gastric ulcerations. A subtotal gastrectomy was performed. On the second post-operative day the patient had mild, recurrent, upper GI bleeding that was treated with transfusion. Endoscopy on the following day demonstrated minimal oozing of blood along the site of anastomosis and a normal esophagus and duodenum. On the fourth post-operative day, the patient had massive upper GI bleeding, became hypotensive, and was taken to the operating room. The stomach remnant was "tensely distended with blood" and "brisk, arterial bleeding" was noted at the gastroesophageal junction. Intraoperative endoscopy revealed a large clot and bleeding from the mid-esophagus. The patient received massive transfusions but continued to bleed at an uncontrollable rate and expired in the operating room.

A dissecting aneurysm of the aortic arch with fistula formation in the esophagus was found on autopsy.

Discussion

The first case of aortoesophageal fistula (AEF) was reported in 1818 by Dubreuil, a French naval surgeon.[1] A 28-year-old soldier had swallowed a 3.3 cm piece of animal rib and after several days developed dysphagia and chest pain, suddenly vomited bright red blood and died. Autopsy revealed the piece of rib to be perforating the esophagus and descending aorta.

A review of 314 cases of esophageal foreign bodies by Barrie et al in 1946, reported the occurrence of AEF in 14 (4.7%) patients.[2] Most of these cases occurred in men who had eaten fish and animal bones. Theories attempting to explain the epidemiology of these cases focused on "poor table manners and an unhealthy tendency to bolt down food".[3] Since 1934 fewer cases have been reported and this has been attributed to dietary changes and/or better table manners.[4] More recently the majority of AEFs from ingested foreign bodies have been reported in children.[5] A myriad of items have been swallowed, including coins, safety pins, needles, detachable aluminum pop tops and twist-tie wires.

Aortoenteric fistulas can be found at any level from the esophagus to the rectum. Approximately 80% occur at the duodenum[6] and are usually associated with a history of abdominal aortic aneurysm repair.[7] In a series of more than 1800 cases of ruptured aortic aneurysms at all sites, approximately 10% ruptured into the esophagus.[8]

The majority of AEFs are caused by thoracic aneurysms. Funk[9] reported the first case in 1918 and subsequently more than 70 have been described. Prior to 1955 most cases of AEF due to aortic aneurysm were caused by syphilis, but atherosclerosis is now considered to be the most common cause. Ten to twenty percent of ruptured thoracic aortic aneurysms result in AEF.[8] Malignant neoplasm, usually esophageal, is the second most common cause of AEF, although rare. By 1896 Knaut[10] had found 34 cases in the literature with several reports following. Aortoesophageal fistulas have also arisen as a complication of surgical resection of thoracic aorta or esophagus, usually due to leakage of gastric contents from the site of anastomoses, which may result in abscess formation with subsequent erosion of the aorta[11-17]. Esophageal intubation with a Celestin tube in obstructing esophageal carcinoma and irradiation of esophageal tumors have also been associated with AEF[18,19]. Approximately 0-1.5% of AEFs are caused by chronic gastroesophageal reflux.[20] Cases of penetrating peptic ulcers of the esophagus,[15,21] esophageal ulceration from achalasia[22], and mediastinitis resulting from ulcerating esophagitis[19] have been reported. Other rare causes of AEF include: placement of a prosthetic heart valve[23]; caustic injury[18,24] eg, lye ingestion[25]; and iatrogenic causes following aortic surgical procedures for vascular rings, aortic coarctation, and patent ductus.[18]

In 1914 Chiari described the clinical triad of the aortoesophageal syndrome: 1) esophageal injury causing midthoracic pain, usually followed by a symptom-free interval, 2) "sentinel" or "herald" hemorrhage, leading to 3) fatal exsanguination in hours to days.[26] The midthoracic pain is attributed to distention, erosion, and dissection of the aortic wall; esophageal compression or perforation with mediastinitis; or tumor involving the aorta

or pleura.[18] The "sentinel" hemorrhage is transient, usually recurrent, arterial bleeding that manifests as hematemesis and/or melena. This is often described as the most significant finding of AEF, in that it allows for life-saving surgical intervention if recognized propitiously. The herald bleed is followed by a latent period of hours to days (occasionally even weeks or months) before the final exsanguination occurs.

The common pathogenesis of AEFs, excluding those caused by foreign bodies is thought to be due to occlusion and thrombosis of the vaso vasorum by fibroblasts, inflammatory cells, or neoplastic cells, rather than by direct invasion of the aorta. Damage of the vaso vasorum leads to aortic infarction with eventual dissolution of the aortic wall and erosion in to the esophagus. Periaortic hematoma may partially tamponade the initial hemorrhage, but as aortic necrosis progresses, it results in terminal exsanguination. Hypotension, vasoconstriction secondary to hypovolemia, and a tamponade effect caused by increased intraluminal esophageal pressure, have also been purported to contribute to the pathogenesis of the latent period.[27,28]

Puncture of the aorta immediately upon ingestion of a foreign body is rare;[4] more frequently there is a delay before the final hemorrhage. Several mechanisms to explain the intermittent bleeding have been suggested. Some investigators propose that the foreign body itself may plug the aortic injury[29], or that the clot may tamponade the aortic defect, resulting in stasis . and temporary closure of the fistula[30]. In a review of 81 cases of AEF due to ingestion of a foreign body, Sloop et al found that most perforations occurred one to 5 cm from the origin of the left subclavian artery, where there is a relative narrowing of the esophagus and where it most closely approximates the aorta.[4] Perforation of the proximal aortic arch and the lower thoracic aorta was observed less frequently.

Surgical intervention offers the only possibility for survival.

To make the diagnosis of AEF, a routine chest x-ray may show widening of the mediastinum or the presence of a foreign body, but more commonly it will fail to show any abnormality. Contrast studies of the esophagus and aorta do not reliably establish the site of fistula formation; however, they often confirm the presence of an esophageal or aortic lesion.[18,28,31,32] Computed tomography with contrast defines aortic and esophageal anatomy, but is not the primary test in acute gastrointestinal hemorrhage. A barium study may outline a luminal abnormality if hematoma, foreign body, or tumor is present, but similarly, it is rarely the procedure of choice in the acute presentation of gastrointestinal bleeding. Aortography may be diagnostic if the fistula is demonstrated by contrast leak from an aneurysm, however active bleeding is rarely seen.[7] The fistulous tract may not be visualized if it is occluded by a blood clot or if the bleeding is inactive at the time of the procedure.[11,18,28,33] "Even when the contrast extravasates into the esophagus, it may run off quickly from the site of the fistula to pool in the proximal or distal esophagus".[33] Thus angiography is considered to be a less sensitive indicator of AEF when compared to barium studies.[11,18,28,33,34]

The role of endoscopy in suspected AEF is uncertain.[7] The risk of precipitating massive hemorrhage[28,35] has made the use of endoscopy controversial. Some authors recommend that it be done only intraoperatively where thoracotomy can easily be performed if necessary,[31,36] whereas others have found that it is safe as long as a biopsy is not obtained.[7] Although gastroscopy has been found to have a diagnostic sensitivity of only 38% in AEF,[37] it can often detect more common causes of bleeding. Endoscopy is usually the procedure of choice in acute upper gastrointestinal bleeding, and has usually been performed before AEF is suspected.

Although the first case of AEF was reported in 1818[1], successful treatment was only described in 1961 by Tanieski[38] and in a few cases subsequently[32,35,39-41]. Surgical intervention offers the only possibility for survival. The Sengstaken-Blakemore tube has been used as a temporary measure to prevent exsanguination[41,42] but must be followed by surgery.

Summary

Survival in AEF is rare because the diagnosis of this uncommon entity is not always suspected, and few patients survive despite aggressive but often late intervention. We present this case to increase awareness of AEF, which although rare, does occur and should be suspected in any patient who presents with midthoracic pain or dysphagia and herald bleeding.

References

[1] Dubreuil. Observation sur la perforation de l'oesophage et de l'aorte thoracique par une portion d'os avale: avec des reflexions. J Univ Sci. Med. 1818; 9:357-363.

[2] Barrie, HW, Townrow V. Perforation of the aorta by a foreign body in the esophagus. J. Laryngol. Otol. 1946; 61:38-42.

[3] Grey, TC, Mittleman RE, Wetli CV, Horowitz S. Aortoesophageal fistula a and sudden death: A report of two cases and review of the literature. The Am J Forensic Med Pathol 1988; 9(1):19-22.

[4] Sloop RD, Thompson JC. Aortoesophageal fistula: Report of a case and review of the literature. Gastroenterology 1967; 53:768-77.

[5] Salam EL, Bargay A, Foad H, Fathi A. Esophageal fistulae caused by swallowed foreign bodies. J. Laryngol. Otol. 1969; 83:251-9.

[6] Pressler V, MacNamara JJ. Thoracic aortic aneurysms. Natural history and treatment. J Thorac Cardivasc Surg 1980; 79:489-498.

[7] Sosnowik D, Greenberg R, Bank S, Graver M. Aortoesophageal fistula: early and the late endoscopic features. Am J Gastroenterol 1988; 83(12):1401-4.

[8] Hooper WL. Haematemesis and melena due to rupture of a saccular aneurysm of the aorta into the oesophagus. Postgrad Med J 1962; 38:297.

[9] Funk EH. Aortic aneurysm with esophageal rupture. Med Clin North Am 1918; 2:795-802.

[10] Knaut B. Uber die durch speiserohrenkrebs bedingten perforation der benachbarten Blutbahen, nebst einer Beobachtnung von primarer Oesophagus dilatation und von Leukoplakia esophagi. Inaugural Dissertation, Berlin, Vogt, 1896.

[11] Tierney LM Jr, Wall SD, Jacobs RA. Aortoesophageal fistula after perigraft abscess with characteristic CT findings. J Clin Gastroenterol 1984; 6:535-7.

[12] Seymour EQ. Aortoesophageal fistula as a complication of aortic prosthetic graft. A J R 1978; 131:160-1.

[13] Sturg BS, Saltzman DA, Feldman MI, et al. Aortoesophagea fistula. Cardiovasc Res Cent Bull 1979; 18:34-8.

[14] Merendino KA, Emerson EC. Aortoesophagogastric fistula, and unusual complication of esophagogastrostomy performed under the aortic arch following esophageal resection for carcinoma. A report of two cases. J Thorac Surg 1950; 19:405-411.

[15] Maguire WC, Mitchell N. Perforation of the aorta by acid gastric contents at site of gastroesophagostomy. Surgery 1947; 22:842-4.

[16] LeRoux BT. Aortic erosion complication oesophagogastrectomy. Br J Surg 1961; 49:271-7.

[17] Ullmann AS, Shier KJ, Horn RC. Aortoesophageal fistula: An unusual complication of esophagogastrostomy following resection for carcinoma of the esophagus. Can Med Assoc J 1961; 85:27-31.

[18] Carter R, Mulder GA, Snyder EN, et al. Aortoesophageal fistula. Am J Surg 1978; 136:26-30.

[19] Marcelo FB, Jr. Aortoesophageal fistula: An unusual complication of esophagogastrostomy following resection for carcinoma of the esophagus. Can Med Assoc J 1961; 85:27-31.

[20] Edwards BS, Edwards WD, Connallly DC, et al.. Arterial-esophageal fistulae developing in patients with anomalies of the aortic arch system. Chest 1984; 86:732-5.

[21] Lefkowitz M, Elas LJ II, Levine RJ. Candida infection complicating peptic esophageal ulcer. infection in an aortic-esophageal fistula. Arch int Med 1964; 113:672-5.

[22] MacPherson DJ, Thompson WR. Aorticesophageal fistula secondary to achalasis accompanied by megaesophagus and esophageal ulceration. Medical intelligence 1958; 259:1027.

[23] Kittle CF. Aortoesophageal fistula: A late complication following insertion of a Hufnagel valve. J Thorac Surg 1985; 36:44-8.

[24] Couves CM, Howard JM, Amerson JR. Fatal perforation of the thoracic aorta by a gastric ulcer. A late complication of esophageal resection. Am J Surg 1958; 95:878-81.

[25] Ottosson A. Late aortic rupture after lye ingestion. Arch Toxicol 1976; 47:59-62.

[26] Chiari H. Uber premdkorperveletzund des oesophagus mit aortenperforation. Beri Klin Wochenschr 1914; 15:7-9.

[27] Henry WJ, Miscall L. Aorto-esophageal fistula. J Thorac Cardiovasc Surg 1960; 39:258-62.

[28] Baron RL, Koehler RE, Gutierrez FR, et al. Clinical and radiographic manifestations of aortoesophageal fistulas. Radiology 1981; 141:599-605.

[29] Taylor CJ. Case of perforating wound of oesophagus and aorta by fish bone. Brit Med J 1913; 1:1207-8.

[30] Ferguson MJ, Arden MJ. Gastrointestinal hemorrhage secondary to rupture of aorta. A review of four duodenal and three esophageal cases. Arch Intern Med 1966; 117:133-40.

[31] Lui RC, Johnson FE, Horovitz JH, et al. Aortoesophageal fistula: Case report and literature review. J Vasc Surg 1987; 6(4):379-82.

[32] Snyder DM, Crawford ES. Successful treatment of primary aortoesophageal fistula resulting from aortic aneurysm. J Thorac Cardiovasc Surg 1983; 85:457-63.

[33] Khawaja FI,, Varindani MK. Aortoesophageal fistula: Review of clinical radiographic, and endoscopic features. J Clin Gastroenterol 1987; 9(3):342-4.

[34] Myers HS, Silber W. Oesophageal bleeding from aortoesophageal fistula due to aortic aneurysm. S Afr Med J 1983; 63:124-7.

[35] Ctercteko G, Mok CK. Aorto-esophageal fistula induced by a foreign body. The first recorded survival. J Thorac Cardiovasc Surg 1980; 80:233-5.

[36] McFaddin DW, Dang C. Management of aortoesophageal fistula. Am Surg 1985; 51:548-550.

[37] Perdue GD, Smith RB, Ansley JD, et al. Impending aortoesophageal hemorrhage: The effect of early recognition on improved outcome. Ann Surg 1980; 192:237-43.

[38] Taniewski J. (Hemorrhage from the aorta caused by a foreign body). Otolaryngol Pol 1961; 15:367.

[39] Yonago RHL Iben AB, Mark JB. Aortic bypass in the management of aortoesophageal fistula. Ann Thorac Surg 1969; 7:235-7.

[40] Patel NM, Sangchantr WS. Aortoesophageal fistula and sentinel hemorrhage. I M J 1985; 167(1):58-9.

[41] Wasmuth H, Verhage CC. Hematemesis due to fistula between aorta and esophagus and later to fistula between left common carotid artery and esophagus. Acta Chir Scand 1989; 155:67-9.

[42] Laforet EG. Aortoesophageal fistula due to foreign body. Milit Med 1975; 140:278-80.

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Urological Injury and Assessment in Patients with Pelvic Fractures

David M. Albala, MD
John R. Richardson, Jr., MD
John A. Heaney, MD, FRCSI

Pelvic fractures account for approximately 3% of all bony fractures.

Patients sustaining pelvic fractures secondary to trauma are known to be at risk for an associated bladder and/or urethral injury. Posterior urethral rupture is by far the most important of these injuries because of its high incidence of serious complications: namely stricture, impotence, and incontinence. In a review by Cass,[1] the incidence of injury to the lower urinary tract in patients with pelvic fractures was 7 to 25%. When there is trauma to multiple organ systems and patient care is segmented across various surgical disciplines, urological injuries initially may not be detected unless a high index of suspicion exists. If not promptly recognized and treated, such injuries will likely cause severe late complications that may prove exceedingly difficult to correct.

Such problems prompted us to review the evaluation of patients at our institution who were found to have traumatic pelvic fractures. Our retrospective review determined the incidence of clinically significant urological injury and the clinical and radiographic findings associated with these injuries. We also sought a diagnostic plan for ruling out bladder or urethral injury in such individuals.

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Methods

A detailed review was made of 160 patients with major pelvic fractures seen at our hospital from July 1, 1983 to July 1, 1988. The records of each patient were evaluated as to age, sex, cause of injury, type of fracture, and urological symptoms and signs. Urological diagnostic evaluation was done for every patient as described by Porter.[2] The radiographic records of each patient were evaluated to determine the type of injury and its possible relationship to the urinary tract.

The pelvic fractures were classified using the method of Trunkey *et al*:[3]

Type I
Comminuted (crush) injuries;
Three or more major components involved (rami, ilium, acetabulum, sacrum)

Type II
Unstable (require immobilization or traction);
A: Diametric fractures with cranial displacement of hemipelvis
B: Diametric fractures, undisplaced
C: Open-book (sprung) pelvis
D: Acetabular fractures

ABBREVIATIONS USED:
RBC/HPF: red blood cells per high power field

Type III
Stable (immobilization usually unnecessary except for symptomatic relief);
A: Isolated fractures
B: Fracture of the pubic rami

Results

Age and Sex. The average patient age was 32 years (range 7 to 84 years). Sixty-three percent of the patients were male and 37% were female.

Etiology of Injury. Motor vehicle and vehicular-pedestrian accidents accounted for 77% of the injuries, and male patients were most frequently the driver of the vehicle or the injured pedestrian; female patients were most frequently passengers in the vehicles (Table 1).

Type of Urological Injury. Fourteen patients sustained a urological injury (Table 2). Eight patients had only a ruptured bladder (5 extraperitoneal, 2 intraperitoneal, and 1 combined extraperitoneal/intraperitoneal injury). There were 5 male patients with a ruptured urethra and 1 male patient with an extraperitoneal bladder rupture and a urethral rupture.

Diagnostic Signs. One hundred and nineteen of the 143 patients who had urinalysis had hematuria, and 24 patients had fewer than 10 red blood cells per high power field (RBC/HPF) on urinalysis (Table 3). Of these 24 patients, 19 had a Type II or III pelvic fracture and 5 had severe Type III injuries. Only one of these patients had urinary tract damage.

Of the 8 patients with bladder rupture, 6 were associated with gross hematuria and 2 were associated with

Table 1 **Etiology of Injury**

	Number of Patients	Percent male Patients
Motor vehicle accident (driver)	69	71
Motor vehicle accident (passenger)	28	32
Pedestrian accident	26	64
Motorcycle accident (driver)	12	100
Motorcycle accident (passenger)	3	0
Falls	18	55
Bicycle accident	1	100
Waterskiing accident	1	100
Snowmobile accident	2	50

Table 2 **Type of Urological Injury**

	Number of Patients
Bladder rupture	
Intraperitoneal	2
Extraperitoneal	5
Combined	1
Urethral rupture	5
Bladder rupture (extraperitoneal) and urethral rupture	1
Total number of patients	14
Male 11	
Female 3	

Table 3 **Urinalysis Associated with Pelvic Fractures**

	Male Patients	Female Patients	Total
Gross hematuria	28	13	41
Microscopic hematuria[a]	53	25	78
<10 RBC/HBF[b]	10	14	24
Urinalysis not available	10	7	17
TOTALS	101	59	160

[a]Microscopic hematuria > 10 RBC/HBF
[b]RBC/HPF: Red blood cells per hgh power field
(In our classification < 10 RBC/HPF is not significant hematuria; therefore there are 3 groups: 1) Gross hematuria; 2) Microscopic hematuria; and 3 <10 RBC/HPF

Table 4 **Physical Signs Associated with Urethral Rupture in Men with Pelvic Fractures** (N=6)

Sign	Number
Blood at urethral meatus	4*
Elevated prostate	1
Scrotal/perineal swelling	1
No physical findings	1

*Blood at urethral meatus and an elevated prostate gland occurred together in one patient.

Table 5 **Type of Pelvic Fracture* and Urological Injury**

Type		Number
I	Comminuted (crush) injuries	8
II	Unstable	
	(A) Diametric fractures with displacement	3
	(B) Diametric fractures, undisplaced	0
	(C) Open-book (sprung) pelvis	0
	(D) Acetabular fractures	1
III	Stable	
	(A) Isolated fractures	0
	(B) Fractures of pubic rami	2

*Classification according to Trunkey *et al:* Management of pelvic fractures in blunt trauma injuries. J Trauma 14:912, 1974.

microscopic hematuria (>10 RBC/HPF). Five patients had ruptured urethras with physical signs of blood at the urethral meatus, an elevated prostate gland, or scrotal/perineal swelling. In one patient there were no physical findings associated with the urethral rupture (Table 4).

Type of Pelvic Fracture. Fourteen patients had urological injuries. In all cases of urethral rupture pubic rami fractures were present. Any type of pelvic fracture can be associated with a urological injury, although the more severe the fracture the higher the incidence of injury (Table 5).

Treatment. Both patients with intraperitoneal rupture, the 3 with extraperitoneal, and 1 with combined intraperitoneal/extraperitoneal bladder rupture were handled by formal surgical repair and catheter drainage for at least 10 days with excellent results. Two patients with extraperitoneal bladder ruptures were managed with 12 days of urethral catheter drainage alone. One patient died in the immediate postoperative period of causes unrelated to the bladder injury (myocardial infarction).

All 5 patients with a urethral injury were treated with suprapubic drainage only for 3 to 6 months. Two patients with a partial tear were able to void by 6 weeks and 8 weeks after the accident, respectively, and required no further treatment. The other 3 patients underwent successful one-stage urethroplasty 6 months later.

Discussion

Pelvic fractures account for approximately 3% of all bony fractures, and about 1 in 1,000 hospital admissions is attributable to this injury.[4] During the first half of this century most pelvic fractures associated with lower urinary tract injuries were caused by industrial accidents, occurred in male patients, and had a higher incidence of urethral injury than today. Prather and Kaiser[5] reviewed 11 series published from 1878 to 1946, and Wilkinson[6] reviewed 10 series published from 1909 to 1946. They found that between 0.7 to 25% of the patients with pelvic fractures had lower urinary tract rupture, and approximately 66% of these were urethral injuries.

Our overall injury rate was 9%, with 5% of those caused by bladder rupture and 4% by urethral rupture. Motor vehicle accidents accounted for the majority of these injuries, and in similar circumstances, males were injured three times more frequently than females. Less common causes were falls from heights, sports injuries, or industrial crush injuries. These findings

correlate well with the reports in the literature as to the expected modern incidence and distribution.

Bladder rupture occurs in approximately 5 to 10% of patients with pelvic fractures.

Bladder rupture occurs in approximately 5 to 10% of patients with pelvic fractures.[7] The injury may be extraperitoneal (50 to 85%), intraperitoneal (15 to 45%), or, rarely, both (0 to 12%).[7] Classically, an intraperitoneal rupture consists of a tear in the dome of the bladder, which is covered by peritoneum. This injury occurs when the bladder is full and a blow is delivered to the lower abdomen. The intravesical pressure is elevated and the bladder ruptures at its weakest point. Extraperitoneal bladder rupture was believed to result exclusively from direct penetration of the bladder wall by a bony spicule or by disruption of the ligamentous attachments between the bladder and pelvis.[8] However, Carroll and McAninch[9] noted only 35% of the bladder injuries in their series to be on the same side as the pelvic fracture. Cass and Luxenberg[7] reported similar findings and felt that with the bladder empty severe lower abdominal trauma may cause a bursting or extraperitoneal penetration similar to that which occurs when the bladder is full.[7]

The incidence of urethral injuries associated with pelvic fractures in men has been reported as 1.4 to 11%.[10] Urethral injuries are rare in female trauma patients because the female urethra is relatively mobile and lacks attachment to the pubis.[10] Pokorny *et al*[11] have described four mechanisms responsible for urethral injury in pelvic trauma: three involve shearing forces generated at the time of pelvic disruption and one involves direct laceration by a displaced bony spicule, which is believed to be a rare occurrence.

All patients with pelvic fracture should be suspected of having a urologic injury. Gross hematuria or blood at the urethral meatus is the cardinal sign of potentially serious urological injury and was noted in 9 of our patients (64%) with urethral or bladder disruption. In two patients microscopic hematuria was associated with bladder rupture. An inability to urinate may be seen in patients with a bladder perforation or a urethral injury. However, more common causes in the trauma victim include an empty bladder or simply an inability to void on command from pain or shock from the fractured pelvis.

The classic perineal butterfly hematoma is diagnostic of urethral disruption include blood at the urethral meatus and a high-riding prostate. Low *et al*[12] recommend that all pelvic fractures involving the sacroiliac joint and pubic ram, in addition to those with the physical signs described previously, warrant a retrograde urethrogram before instrumentation. Rectal examination is imperative and may reveal an "absent" prostate, a pelvic hematoma, or more importantly, an associated rectal laceration.

All patients with pelvic fractures should be suspected of having a urologic injury.

Trauma patients with pelvic fractures and suspected urologic injury should not undergo urethral catheterization until the integrity of the urethra has been radiographically demonstrated for fear of converting a partial urethral tear into a complete disruption.[12] A retrograde urethrogram is performed with the patient in the oblique position with the penis stretched perpendicular to the femur. With the patient in the oblique position the shortening of the bulbous urethra is avoided and the entire urethra may be examined adequately.[13] If the urethra is normal, a catheter may then be advanced into the bladder and a cystogram can be performed. If a catheter has been inserted without first examining the urethral anatomy, a urethrogram can be obtained by placing a large angiocatheter alongside the catheter and injecting contrast while compressing the urethra around the catheter. Extravasation of contrast medium without filling of the bladder is diagnostic of a complete urethral disruption. Partial urethral disruption is diagnosed in the presence of extravasation with partial filling of the bladder.[13]

Cystograms should be performed on all patients with suspected bladder injuries by injecting 300 to 500 ml of dilute contrast material via a urethral catheter. With the bladder distended, a radiograph is then

obtained with the patient in the anteroposterior position. The contrast material is drained from the bladder and a post-drainage film is then obtained. Small amounts of extravasation not visible behind a contrast-filled bladder will be readily identifiable on a post-drainage film.[14]

Rectal examination is imperative and may reveal an absent prostate, a pelvic hematoma, or more importantly, an associated rectal laceration.

Our present diagnostic evaluation of the lower urinary tract with a pelvic fracture is outlined (Fig. 1). Voided urines can almost always be obtained in the female patients, since (as discussed earlier) injuries to the urethra after pelvic fracture are almost nonexistent. Cystograms should be obtained in patients with gross or microscopic hematuria (>10 RBC/HPF). Males with perineal or scrotal hematomas, blood at the urethral meatus, high-riding prostates, or pelvic fractures involving the sacroiliac joint and pubic rami warrant a retrograde urethrogram before instrumentation. In no case is the passage of a Foley catheter warranted as a diagnostic step to determine urethral injury.

Conclusion

Thorough, well-planned urologic evaluation of the patient with pelvic trauma is essential to prevent potentially crippling and occasionally fatal sequelae of thee injuries. The urologist is a vital member of the treatment team and must be prepared to manage the initial and reconstructive surgery necessary for these patients.

References

1 Cass AS: Bladder trauma in the multiple injured patient. J Urol 115:667, 1976.

2 Porter JE: Urologic injuries. Surg Clin N Am 57:77, 1977.

3 Trunkey DD, Chapman MW, Lim RC, and Dunphy JE: Management of pelvic fractures in blunt trauma. J Trauma 14:912, 1974.

4 Spirnale JP: Pelvic fracture and injury to the lower urinary tract. Surg Clin N Am 68:1057, 1988.

5 Prather GC and Kaiser TF: "The bladder in fracture of the bony pelvis: The significance of a "tear drop bladder" as shown on cystogram. J Urol 63:1019, 1950.

6 Wilkinson FC: Rupture of the posterior urethra; with a review of 12 cases. Lancet 1:1125, 1961.

7 Cass AS and Luxenberg M: Features of 164 bladder ruptures. J Urol 138:743, 1987

8 Brosman SA and Fay R: Diagnosis and management of bladder trauma. J Trauma 13:929, 1973

9 Carroll PR, McAninch JW. Major bladder trauma: Mechanisms of injury and a unified method of diagnosis and repair. J Urol 132:254, 1984.

10 Colapiuto V: Trauma to the pelvis: Urethral injury. Clin Ortho 151:46, 1980

11 Pokorny M, Pontes JE, and Pierce JM: Urological injuries associated with pelvic trauma. J Urol 121:455, 1979.

12 Low MA, Mason JT, Luna, GK, Maier RV, Copass MK, and Berger, RE: Risk factors for urethral injuries in men with traumatic pelvic fractures. J Urol 140:506, 1988.

13 Sandler CM, Harris JH, Carriere JN, and Toombs BD: Posterior urethral injuries after pelvic fractures. Am J Roentgen 137:1233, 1981.

14 Carrol PR, McAninch JW. Major bladder trauma: The accuracy of cystography. J Uro 130:887, 1983.

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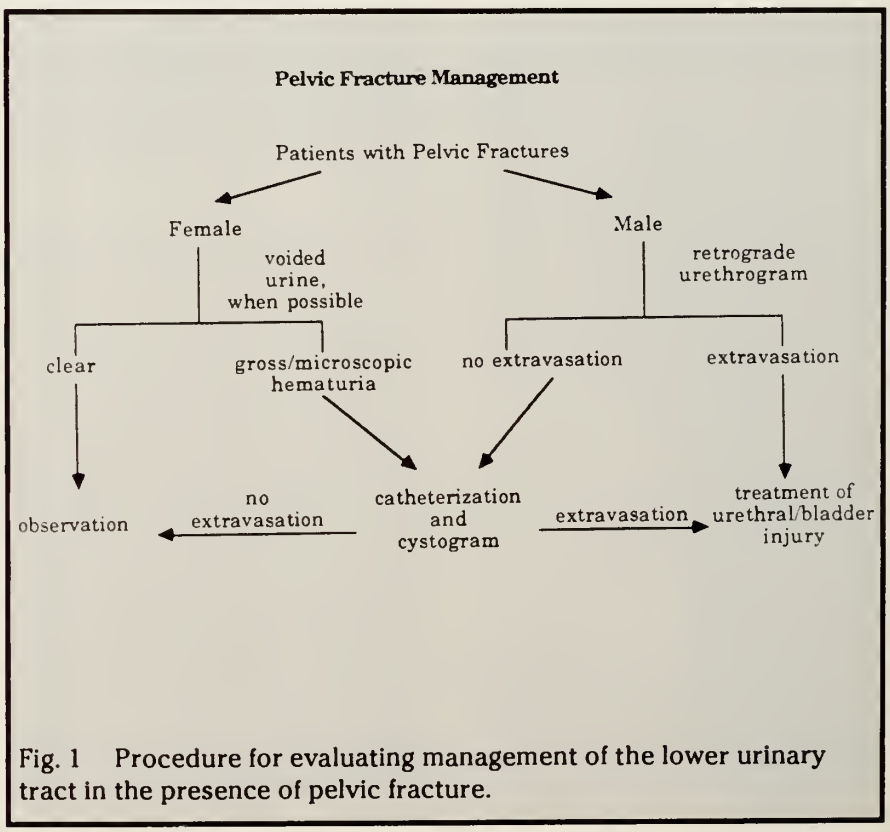

Fig. 1 Procedure for evaluating management of the lower urinary tract in the presence of pelvic fracture.

Diagnosis and Treatment of Bladder Rupture: Characteristics of 42 Cases

David M. Albala, MD
John R. Richardson, Jr., MD

Trauma to the urinary bladder may result from (1) blunt impact to the lower abdomen or pelvis . . . (2) penetrating injuries . . . or (3) intraoperative injury to the bladder during surgical procedures . . .

Bladder rupture is an uncommon injury, but its presence usually implies the risk of significant morbidity and sometimes mortality. The bladder, located within the pelvic cavity, is protected by the bony pelvis and cushioned by perivesical fat, the sigmoid colon, and the levator muscle sling. It is an extraperitoneal structure, except for the uretheral hiatus, the dome, and the posterior surface superior to the trigone. This superior portion, covered by visceral peritoneum, protrudes into the peritoneal cavity during filling and lacks the support of the surrounding tissues. It is susceptible both to direct penetrating injury and to hydraulic rupture resulting from pelvic or peritoneal impact.

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Trauma to the urinary bladder may result from (1) blunt impact to the lower abdomen or pelvis (eg, that caused by automobile accidents, falls, contact tact sports, and crush injuries); (2) penetrating injuries (eg, those from gunshot and knife wounds); or (3) intraoperative injury to the bladder during surgical procedures involving adjacent organs. These injuries can cause bladder contusion, intraperitoneal or extraperitoneal bladder rupture, or a combination of these conditions. The type of injury sustained usually can be predicted from the mode of injury and the finding on initial physical and radiologic examinations. This study reviews the characteristics of 42 patients with bladder rupture and outlines our present methods of diagnosis and treatment.

Patients and Methods

A detailed review of all patient admissions to our hospital from January 1969 to August 1987 disclosed 42 patients with a final diagnosis of bladder rupture. We noted age, sex, cause of injury, presence of pelvic fracture, associated injuries, urologic signs and symptoms, treatment, attendant complications, and length of hospital stay for each patient.

In all cases the presence of macroscopic or microscopic hematuria was an indication for immediate radiologic evaluation of the urinary tract. Radiologic evaluation consisted of a retrograde cystogram with a drainage film. In selected patients an intravenous pyelogram was performed to evaluate the upper urinary tracts. If blood was present at the external urinary meatus a retrograde urethrogram was performed before passing the urethral catheter. If extravasation was seen on the cystogram, exploration of the bladder with repair of the rupture and drainage of the bladder by suprapubic cystostomy and/or urethral catheter with drainage of the perivesical space were

performed. Since 1980, urethral catheter drainage without exploration repair of the bladder has been used in selected patients with extraperitoneal rupture of the bladder. Follow-up cystograms were obtained 8 to 12 days after catheter placement. If no extravasation was noted the catheter was removed, unless it was required for nursing care or for associated urethral injury.

Results

Forty-two patients with bladder rupture were diagnosed and treated in the 18 years under review. Of this group, 19 (45%) were male and 23 (55%) were female. Ages ranged from 6 to 87 years; 35 patients (83%) were less than 45 years old.

Location and Mechanism

Blunt external trauma was responsible for 40 injuries and consisted of motor vehicle accidents in 34, falls in 5, and a crush injury in 1. Of these patients, there were 27 (68%) with extraperitoneal ruptures, 11 (27%) with intraperitoneal ruptures, and 2 (5%) with combined intraperitoneal and extraperitoneal ruptures. The two other patients sustained a penetrating injury from a gunshot wound and an iatrogenic injury that occurred during gynecologic surgery, respectively. Both patients had associated extraperitoneal injury. All intraperitoneal ruptures were due to blunt trauma and consisted of a tear in the dome of the bladder.

Associated Injuries

Associated injuries were present in 37 patients (88%). A fractured pelvis was the most common associated injury, presenting 30 patients (71%). Pubic rami fractures were found in 37% of the patients with extraperitoneal ruptures, and in 20% of those with intraperitoneal ruptures. There was no significant relationship between the type of pelvic fracture and the specific type of bladder rupture (Table 1). However, the data suggest that with pubic rami fractures, one needs to have a higher index of suspicion of bladder injury than when other fracture locations are considered.

There was no significant relationship between the type of pelvic fracture and the specific type of bladder rupture.

Associated genitourinary injuries (ie, renal contusion, urethral rupture, etc.) were present in two patients with intraperitoneal rupture and in seven patients with extraperitoneal rupture. Urethral rupture was the most common genitourinary injury and occurred only in patients with associated pelvic fractures (Table 2).

Clinical Presentation

Hematuria (macroscopic or microscopic) was present in 25 of the 29 patients (90%) with extraperitoneal ruptures, in 9 of the 11 patients (82%) with intraperitoneal ruptures; and in both patients having combined intraperitoneal and extraperitoneal ruptures (Table 3). Gross hematuria occurred in 36 patients (86%), while microscopic hematuria was seen in 6 patients (14%). Abdominal tenderness was present in 29 patients (69%). When first examined, 10 patients (24%) were in shock, defined here as systolic blood pressure less than 90 mm Hg.

Treatment and Outcome

Surgical repair was performed in all patients with intraperitoneal rupture and combined in-

Table 1 **Pelvic Fracture Associated with Bladder Rupture** (N=30)

Type of Pelvic Fracture		Site of Rupture: Intraperitoneal	Extraperitoneal	Combined
I	Comminuted (>3 parts)	1 (3%)	4(13%)	2 (7%)
II A/II B	Anterior/Posterior	2 (7%)	1 (3%)	0
II C	Symphyseal Separation	0	0	0
II D	Acetabulum	2 (7%)	1 (3%)	0
III A	Isolated (eg, Iliac Crest)	0	0	0
III B	Pubic Rami	6 (20%)	11 (37%)	0
	TOTALS	11 (37%)	17 (56%)	2 (7%)

traperitoneal and extraperitoneal rupture. Twenty-one patients with extraperitoneal rupture (72%) had surgery, while nonoperative (conservative) treatment (antibiotics and catheter only) was used in 8 patients (28%). There were no complications associated with conservative therapy. There were two deaths in the study group –one from a myocardial infarction on the seventh hospital day and the other from sepsis 46 days after surgery (Table 4).

Length of hospitalization depended on the site of rupture and associated injuries. The largest group of patients (14) remained in the hospital between 30 and 40 days. Those patients with other injuries (eg, abdominal injuries, pelvic fractures) usually required longer hospital stays.

Discussion

Three major categories of bladder injury are recognized: contusion, extraperitoneal rupture, and intraperitoneal rupture. The type of bladder injury sustained varies according to the degree and mechanism of injury, and the amount of urine in the bladder at the time of trauma. The pelvic location of the empty adult bladder usually provides sufficient protection from traumatic injury. Major bladder trauma accounts for less than 2% of abdominal injuries requiring surgical repair or exploration.[1] However, when the bladder is distended or the pelvis is fractured, the normal protective influences of the intact pelvic ring are lost and bladder injury is relatively common.[2] Most blunt injuries of the bladder occur in motor vehicle accidents; a small number are caused by falls or direct blows to the abdomen.

Bladder contusion is associated with hematoma formation in the perivesical space and with gross hematuria without signs of urinary extravasation; it is the most common type of bladder injury and is usually caused by blunt trauma. The cystographic appearance is that of an intact bladder deformed by a pelvic hematoma (tear-drop deformity). Observation of the patient's condition is the only treatment necessary in most of these patients. Those patients who are immobilized and unable to void while lying in bed may require catheter drainage.

The type of bladder injury sustained varies according to the degree and mechanism of injury, and the amount of urine in the bladder at the time of trauma.

Extraperitoneal rupture occurs most commonly in association with pelvic fractures.[3] Penetrating injuries to the perineum, groin, or lower abdomen can produce the same radiographic and clinical abnormalities. Bladder perforation is usually the result of a direct laceration of the bladder wall by bony spicules[3] or by disruption of the ligamentous attachment between the bladder and the pelvis in conjunction with the pelvic fracture.

Wolk and associates have reported a burst type of extraperitoneal bladder rupture in three patients with extraperitoneal rupture in the absence of pelvic girdle disruption.[4] In another study, only 35% of the extraperitoneal ruptures occurred in the area of the pelvic fracture.[1]

Table 2 **Injuries Associated with Bladder Rupture**

	Site of Rupture		
	Intraperitoneal	Extraperitoneal	Combined
Number of Patients	11	29	2
Number with Associated Injury (other than pelvic fracture)	10	22	2
Genitourinary Injury:			
Renal Contusion	1	2	0
Urethral Rupture	1	3	1
Penile/Scrotal Contusion	0	2	0
Severe Head Injury	2	2	0
Rupture, Abdominal Organ	3	11	1
Other Fractures	7	13	1

Table 3	Clinical Presentations	
	Number of Patients	(%)
Gross Hematuria	36	(86)
9/11 Intraperitoneal		
25/29 Extraperitoneal		
2/2 Combined		
Abdominal Tenderness	29	(69)
Open Wound	11	(26)
Shock	10	(24)
(blood pressure < 90 mm Hg)		

Table 4	Treatment and Outcome		
	Site of Rupture		
	Intraperitoneal	Extraperitoneal	Combined
Surgery (Suture and Catheter)	11	21	2
Urethral Catheter Only	0	8	0
Deaths	0	2	0

Extraperitoneal rupture can occur in a fashion similar to intraperitoneal rupture, ie, by a compression or burst type of injury, and this unexpected pattern occurs as a result of local factors, such as the site and severity of injury, and the extent of bladder distention. In such patients, a pelvic fracture might be coincidental instead of causative. In our series, the site of the extraperitoneal rupture was correlated with the site of the pelvic fracture in 13 patients. In the 4 other patients with pelvic fracture the site of rupture was opposite the fracture; the extraperitoneal rupture presumably resulted from a compression injury.

Intraperitoneal rupture is most often a result of indirect injury to a full bladder, rather than a direct puncture by a bony fragment. The patient usually sustains a blow to the lower abdomen with the force directed equally toward all surfaces of the bladder. The increased pressure leads to a rupture at the dome, the bladder's weakest point. The bladder urine is expressed into the peritoneal cavity, and the continued extravasation of urine may lead to generalized peritonitis in 24 to 48 hours. Therefore, early detection of this injury is crucial. In our series, 11 patients (26%) with intraperitoneal rupture were diagnosed and treated immediately.

Most blunt injuries of the bladder occur in motor vehicle accidents; a small number are caused by falls or direct blows to the abdomen.

The hallmark of bladder rupture is the presence of hematuria after trauma. Some patients will experience suprapubic pain, while others may be unable to void. Most patients will have pelvic fractures and other injuries, which will be potential causes of nonspecific pain. In our series gross hematuria occurred in 36 patients (86%) while microscopic hematuria was seen in 6 patients (14%). This compares favorably with the Parkland Memorial Hospital series in which 45 of 46 patients with rupture of the bladder had gross hematuria on presentation.[5]

Initial evaluation of the traumatized patient with hematuria should be directed at excluding the presence of urethral injury. Any male patient with a pelvic fracture, perineal or scrotal hematoma, or blood at the urethral meatus, should undergo retrograde urethrography before insertion of a urethral catheter. Once urethral injury has been ruled out and a catheter passed into the bladder, a retrograde cystogram should be obtained by infusing 350 cc of water-soluble contrast material. Anteroposterior views and a drainage film are then obtained. Carroll and McAninch demonstrated that if strict attention is paid to adequate distention of the bladder and to obtaining drainage films, diagnostic retrograde cystography will be highly accurate and diagnostic errors should be rare.[6] After a satisfactory retrograde cystogram is performed, evaluation of the upper urinary tract is completed by means of an intravenous

infusion pyelogram. The cystogram obtained from the intravenous infusion pyelogram does not provide an adequate examination of the bladder. In all of our patients a correct diagnosis could be made expediently by retrograde cystogram.

. . . bladder injury . . . can be precisely diagnosed and, if properly managed, carries virtually no morbidity.

The definitive treatment of bladder trauma did not evolve until the early 1900s, when Evans and Fowler advocated that surgery could reduce the mortality associated with bladder rupture.[7] Until that time, bladder wounds were invariably fatal. It was not until after World War I that routine use of suprapubic cystostomy drainage became accepted. Since then, the standard treatment of bladder rupture has been operative repair with drainage of the bladder and perivesical space.

Conservative therapy for patients with extraperitoneal bladder rupture has been advocated only recently. In 1974, Mulkey and Witherington[8] treated eight patients using only catheter drainage, while Richardson and Leadbetter[9] reported similar results for two patients with extraperitoneal rupture and one patient with intraperitoneal rupture. If this method of therapy is elected, a number of points must be kept in mind:

1. The patient must have proven bladder rupture and not be a candidate for surgical exploration because of other complication injury.

2. Diagnosis must be made early (less than 12 hours after trauma).

3. There should be no history of active urinary infection or complicating genitourinary disease. Broad-spectrum antibiotics should be used for two weeks after the trauma.

4. A large-bore Foley catheter (24 F minimum) should be easily inserted.

5. All patients should be hospitalized for observation and monitoring of vital signs and urinary drainage.

6. If for any reason the patient's condition deteriorates surgical exploration and an open drainage procedure must be performed.

We used urethral catheter drainage and intravenous antibiotics without complications in eight patients who had extraperitoneal ruptures. Hayes and associates managed 16 cases of extraperitoneal bladder rupture secondary to blunt trauma by catheter drainage and no antibiotics.[2] In this series there were no septic complications or abscesses noted. However, Cass managed 18 cases of extraperitoneal rupture with urethral catheter drainage alone and four patients had complications, consisting of hematuria in two, pseudodiverticulum and a bone spicule in one, and sepsis, renal failure, and death in one.[10] While the option of non-operative management of bladder rupture is attractive and simple, complications may arise in some patients managed this way.

In summary, bladder injury today can be precisely diagnosed and, if properly managed, carries virtually no morbidity. Conversely, a disorderly approach will mishandle urethral trauma, miss the commonly associated visceral trauma, and is potentially lethal.

References

[1] Carroll PR, McAninch JW. Major bladder trauma: Mechanisms of injury and a unified method of diagnosis and repair. J Urol 1984; 132:254-257.

[2] Hayes EE, Sandler CM, Corriere JN. Management of the ruptured bladder secondary to blunt abdominal trauma. J Urol 1982; 129:946-947.

[3] Weems WL. Management of genitourinary injuries in patients with pelvic fractures. Ann Sur 1979; 6:717-723.

[4] Wolk DJ, Sandler CM, Corriere JN. Extraperitoneal bladder rupture without pelvic fracture. J Urol 1985; 130:1199-1200.

[5] McConnell JD, Wilkerson MD, Peters PC. Rupture of the bladder. Urol. Clin N Am 1982; 9:293-296.

[6] Carroll PR, McAninch JW. Major bladder trauma: The accuracy of cystography. J Urol. 1983; 130:887-888.

[7] Evans E, Fowler H. Puncture wounds of the bladder. Ann Surg 1905; 42:215-217.

[8] Mulke, AP, Witherington, R. Conservative management of vesical rupture. Urology 1974; 4:426-428.

[9] Richardson JR, Leadbetter GW. Non-operative treatment of the ruptured bladder. J Urol 1975; 114:213-214.

[10] Cass AS. The multiple injured patient with bladder trauma. J Trauma 1984; 24:731-734.

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HEALTH BY NUMBERS

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Cancer of the Cervix Uteri in Rhode Island

About 150 Rhode Island women are diagnosed with cancer of the cervix uteri each year. At the time of diagnosis, two-thirds of the tumors are *in situ*, one third invasive. About 15 women die of the disease each year. These deaths are considered avoidable.[1]

Rhode Island's experience with cervical cancer compares favorably with the experience of other states. Since 1950, age-standardized cervical cancer mortality has been lower in Rhode Island than the United States as a whole. Although this differential has diminished over time, Rhode Island still ranked 41st among the states for cervical cancer mortality in the period 1983-1987 (Figure 1)[2]

Mortality from cancer of the cervix uteri is a greater problem for blacks than whites in the United States,[4] and the situation in Rhode Island is no different (Figure 2). In the period 1975-1985, age-standardized cervical cancer mortality in Rhode Island was three times higher among blacks than whites, about two and a half times higher for the United States as a whole.

The Papanicolaou (Pap) test is responsible for a substantial decline in cervical cancer mortality since its introduction in the early 1950s. In Rhode Island, age-standardized cervical cancer mortality among white women declined from 7.7 per 100,000 in the 1950s to 2.6 per 100,000 in 1987-1988.[3] The Pap test is largely responsible for the large proportion of cases diagnosed *in situ*. Recommendations for the frequency of screening with the Pap test in the United States range from one to three years. The Pap test is relatively inexpensive and is covered by most forms of health insurance under a Rhode Island legislative mandate. The cost of the Pap test is also subsidized in federally funded family planning programs; the fee charged is dependent on income. Nonetheless, 13% of Rhode Island women ages 20-39 have not had a Pap test in the past three years, and 24% of Rhode Island women ages 40-69. Data available for the latter age group reveal differences in the use of the Pap test by income (Figure 3), a possible explanation for high cervical cancer mortality rates among black Rhode Island women.

Rhode Island's year 2000 goal for cervical cancer (adopted from the National Health Promotion and Disease Prevention Objectives) is a 50% cut in mortality, from 2.6 per 100,000 (1988) to 1.3 per 100,000 (2000).[5] Towards this end, the Rhode Island Department of Health is working with physicians and cytology laboratories in Rhode Island to improve the reliability of the Pap test, and with the Rhode Island Division of the American Cancer Society and community leaders, to increase the use of the Pap test among poor and minority women. Primary care practitioners throughout the state can help by re-doubling their efforts to check the screening records of their female patients, especially those of women ages 40 and above, who seem to be at especially high risk of not being screened.

References

[1] Fulton, JP, Stanis D: Cancer Rhode Island 1987. Rhode Island Department of Health, Providence, Rhode Island, 1989.
[2] Centers for Disease Control, by special request from the American Association of Central Cancer Registries.
3 US Environmental Protection Agency and National Cancer Institute, Cancer Mortality Rates and Trends, 1950-1979. US Government Printing Office, Washington, 1983.
4. Gloeckler-Ries LA, Hankey BF, Edwards BK (Eds.): Cancer Statistics Review, 1973-1987. NIH Pub. No. 90-2789. National Cancer Institute.
5. US Public Health Service: Healthy People 2000: National Health Promotion and Disease Prevention Objectives. Review Draft. US Department of Health and Human Services, 1990.

Figure 1. Age-standardized death rates, cancer of the cervix uteri, the United States by state quintiles and Rhode Island, 1983-1987

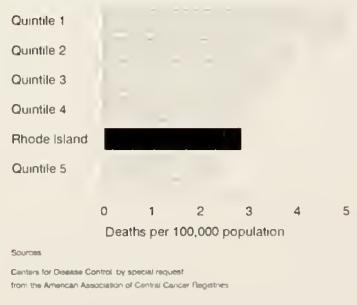


Figure 2. Age-standardized death rates, cancer of the cervix uteri, by race, Rhode Island and the United States, 1975-1985

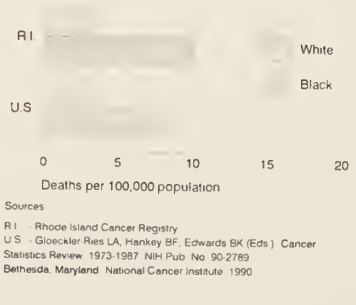
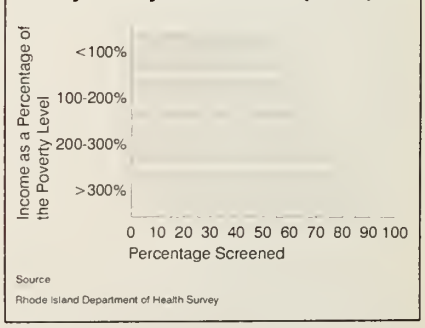


Figure 3. Percentage of women ages 40 and over who had a Pap test in the past three years, by income, 1989 (N=856)



Submitted by the Rhode Island Cancer Registry, John P. Fulton, PhD, Administrator. Health by Numbers is edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD.

Monthly Vital Statistics Report

Provisional Occurrence Data From the Division of Vital Records

H. Denman Scott, MD, MPH
Director of Health

Roberta A. Chevoya
State Registrar

Vital Events	Reporting Period November 1990 Number	12 Months Ending with November 1990 Number	Rates
Live Births	1,313	16,004	16.0*
Deaths	771	9,735	9.8*
Infant deaths	(7)	(127)	7.9#
Neonatal deaths	(6)	(100)	6.2#
Marriages	535	8,186	8.2*
Divorces	251	3,827	3.8*
Induced Terminations	614	7,887	492.8#
Spontaneous Fetal Deaths	76	1,160	72.5#
Under 20 weeks' gestation	(69)	(1,076)	67.2#
20+ weeks' gestation	(7)	(83)	5.2#

*Rates per 1,000 estimated population

#Rates per 1,000 live births

Underlying Cause of Death Category	Reporting Period August 1990 Number (a)	12 Months Ending with August 1990 Number (a)	Rates (b)	YBLL (c)
Disease of the Heart	245	3,496	350.3	5,298.0
Malignant Neoplasms	206	2,449	245.4	6,995.5
Cerebrovascular Diseases	36	589	59.0	713.5
Injuries (Accident, Suicide, Homicide)	35	450	45.1	9.896.0
COPD	21	376	37.7	371.0

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.
(b) Rates per 100,000 current estimated population of 998,000.
(c) Years of Potential Life Lost (YPLL)

NOTE: Total represents vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional total should be analyzed with caution because the numbers may be small and subject to seasonal variation.

THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publication Committee

VOLUME I
NUMBER I

PROVIDENCE, R. I., JANUARY, 1917

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SINGLE COPY, 25 CENTS

THE RHODE ISLAND MEDICAL JOURNAL HERITAGE

Ninety Years Ago

(*The Providence Medical Journal*)
January-March, 1901

The lead editorial advocates more hospitals in Rhode Island. "A city the size of Pawtucket and a large town like Westerly should each have a cottage hospital. Accidents and various emergency cases are now sent a long distance to be cared for when the condition of the patient or the hour of the day or night often make more immediate attention desirable." The editorial continues, "The cottage hospital should be managed, as large hospitals are, by a disinterested board who should have the responsibility of selecting the staff and providing funds for running expenses. A building for such purposes need not be elaborate or expensive; it requires only a few rooms furnished with hospital beds and appliances and a trained nurse in charge." The editorial concludes, "Experience shows that the public listens kindly to appeals for this form of charity and we hope that the physicians of Pawtucket and Westerly will inaugurate the opening year of the new century by an earnest and united effort to supply this urgent need."

A second editorial discusses mosquitoes and malaria and the newly evolved concept that *Anopheles* species mosquitoes carry the plasmodium parasite. This hypothesis, states the editorial, does not serve to explain why unpopulated tracts of swampy land quickly produce malarial fevers in its recent invaders. Since the life of the mosquito is short and there are no human carriers of malaria in the vicinity, it is difficult to understand, says the editorial, where the parasite to infect the first case comes from.

A third editorial discusses the division of authority in the hospital. "Trustees . . . are the ones to care for the business interests of a hospital and as such they should have sole power to regulate its finances, conduct its management, control its superintendent and appoint the professional staff. The staff should have absolute power in the domain of medicine and surgery, and should have such powers so well defined that there would not arise the least possible friction."

The first of the scientific articles, referred to as *Original Communications*, is written by VY Bowditch, MD and discusses State Sanatoria for Tuberculosis. The recent history of the concept of "the segregation of consumptives in special institutions" is reviewed and the inaugural works of Brehmer, Dettweiler, Klebs and Trudeau are discussed. The Massachusetts State Sanitarium, with 175 beds, opened its doors in October of 1898 and has already demonstrated the efficacy of this form of treatment. During the prior year of operation, 39.7% of its patients were listed as "arrested', 52.4% improved, and 7.9% unimproved or died, representing mortality/morbidity statistics which are far better than in the past. The writer concludes with the following statement: "I urge you therefore to press this matter upon your legislature and when you convince them, as I know you can, of the necessity of sanitoria in your own state, be sure that you emphasize the importance of sufficient appropriation to make one institution a model upon which

. . . others may be built when richer experience shall have taught you the wisest methods. I feel confident that Rhode Island will not be far behind her sister state in this march of medical progress."

Yet another paper on the wisdom of segregated facilities for the tubercular patient is presented. The *Journal* then prints some of the formal replies by Rhode Island physicians who attended the meeting. Dr Jay Perkins, a local practitioner, offers the following views: "In fighting this disease (tuberculosis) we have to fight darkness, dirt, foul air and all those things which go to make up unhygienic surroundings at home and in the place of work, and it is necessary to make both the afflicted and the exposed understand the principles and advantages of an enlightened hygienic life. That there is a need for a sanitarium for the use of the consumptives in Rhode Island I believe none of you will question, and yet those of you who practice is among the better classes can not appreciate it fully, as it is a disease which is found in its fullness only under the conditions of dirt, overcrowding and poor ventilation."

A paper by George Keene, MD, of Howard, RI, discusses subcutaneous nutrient injections (hypodermoclysis), which he claims was originated by Catani in the 1865 epidemic of cholera in Naples. He recommends the use of this unusual procedure in cases of severe hemorrhage, ptomaine poisoning, acute delirium and when the digestive system is inoperative. This parenteral technic is distinguished from that of transfusion which the author believes to have been largely unsuccessful, quoting Savonarola's description of the failed attempts in transfusing Pope Innocent VII with the blood of two young males. The subcutaneous infusion is prepared thusly: "The solution used was composed of twelve to fourteen ounces of sterilized water to which, after partial cooling, were added the white of two eggs and thirty grains of common salt. This was filtered through cheese cloth. An ordinary stomach tube and a medium sized aspirating needle were the only instruments employed in giving the infusion."

Fifty Years Ago
(March, 1941)

The lead article is entitled Nutritional Aspects of Pregnancy and is written by Alfred L. Potter, MD. The paper discusses the role of the macro- and micronutrients in sustaining mother and fetus during pregnancy. He concludes, "All ancient and modern authorities agree that the diet of the pregnant woman should be plain, simple, easy of digestion, highly nutritious, and partaken of at regular intervals. In a word, as Lusk said, the diet of pregnancy should be ample in quantity and adequate in quality. We do know better than did Lusk what those words mean."

A.W. Eckstein, MD contributes a case report on swine erysipelas treated with X-ray and sulfanilamide. (Erysipelas was first distinguished from anthrax by Pasteur in 1882. The causative organism, *Erysipelothrix rhusiopathiae* is a gram-positive spore forming bacillus, with strains pathogenic in humans, swine and rodents.) The described patient was a 35-year-old male butcher who had recently lacerated his finger while cutting pork. Within days an erysipeloid lesion involved his arm, neck and face. He was treated by superficial radiation and oral sulfanilamide. He recovered uneventfully.

The lead editorial expresses concern that the incidence rate of cancer in Rhode Island appears to be increasing. By using the discharge data from Rhode Island Hospital, the editorial notes that "In 1929 there were 7,814 discharges from the hospital, 417 with a diagnosis of cancer of some sort. In 1939, of 7,911 discharges, 576 left with a diagnosis of cancer. That shows an increase of 38% in those ten years." The editorial offers no suggestion to account for this apparent rise, but suggests pointedly that the practicing physician ". . . can learn to avoid the gross mistakes in diagnosis that too often lead to delay and make the treatment of malignant disease so hopeless."

Another editorial summarizes the many accomplishments of Dr Charles V. Chapin, Rhode Island's chief health officer for many years as a colleague of Gorgas and Janeway.

The yearly meeting of the New England Health Institute is to be addressed by Dr Thomas Parran, the Surgeon-General of the United States. The three-day seminars will address health issues in national defense.

The minutes of the January 1941 meeting of the Medical Society's Council are published. Dr Lucius Kingman is president, and other members in attendance are Drs Mowry, Partridge, Miller, Burgess, MacLeod, Brackett, Fulton, Hammond, and Wells. Amongst other motions,

the Council adopts the following: ". . . dues of all members of the Rhode Island Medical Society called to the Service of the Flag be remitted for the time of Service."

The 1940 statistics for the Health Department are summarized. The five principal causes of death are: heart disease, cancer, pneumonia, chronic nephritis and cerebral hemorrhage. During 1940 there were two deaths from diphtheria, 57 from pulmonary tuberculosis, two from poliomyelitis, and three from typhoid fever.

This issue of the *Journal* provides its reader with the yearly activities of the Medical Society's Milk Commission specifying butter fat content, total solids and bacterial counts, by month, of the products from each of the local dairy farms offering certified milk.

Twenty Five Years Ago

(March, 1966)

Dr Irving Glickman, Professor and Chairman of Periodontology at Tufts Dental School authors a paper entitled Oral Mucous Membrane Disease. After discussing the histologic features of the oral mucosa and its general pathologic processes, he explores the pathophysiologic basis for bleeding gums and mucosal infections (Vincent's infection, herpetic stomatitis, varicella, moniliasis, and aphtous stomatitis.) The paper then notes that there are oral manifestations of many dermatological disorders, including lichen planus and pemphigus vulgaris. The author concludes by making a plea for the greater use of mucosal biopsy which will thus provide ". . . the clinician a closer understanding of the problem that confronts him and aids him in avoiding ineffective or harmful drugs."

William S. Klutz, MD and Arthur L. Quirk, PhD author a paper entitled The Practicing Physician and the Nuclear Reactor. The article describes the reactor, located in the town of Narragansett, and its capabilities, including: production of isotopes, neutron activation analysis, neutron capture therapy and gamma radiation.

The *Journal* provides its readers with a summary of the Drug Abuse Control Amendments of 1965. These laws pertain to barbiturates, amphetamines and all depressant and stimulant drugs.

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UNDERSTANDING CME

Edited by
Kimberly Allyn,
RIMS CME Coordinator

Janice Miller, M.Ed.,
Director
Office of CME
Brown University

ESSENTIAL #5
EVALUATION
The sponsor shall evaluate the effectiveness of its overall continuing medical education program and of its component activities and use this information in its CME plannings. The sponsor shall:

1) Periodically review the extent to which the sponsor's CME mission is being achieved by its educational activities.

2) Show that these evaluations assess:

a. the extent to which educational objectives are being met;

b. the quality of the instructional process

c. participants' perception of enhanced professional effectiveness.

3) Use evaluation methods which are appropriate and consistent in scope with the educational activity.

4) Demonstrate that evaluation data are used in planning future CME activities.

Used effectively, the evaluation process can be an important tool in determining the success of the CME program. The actual evaluation process is two-fold: evaluation of individual CME activities and evaluation of the overall CME program.

When evaluating individual CME activities, key points should be addressed in the evaluation sheets. Participants should be able to answer whether the stated objectives have been met as well as the effectiveness of the format, quality of the speaker and appropriateness of the facilities. Other indicators of a successful CME activity may be recorded by observing level of participant interaction as well as attention and interest. Evaluation sheets may be distributed to each participant or a handful of respondents may be selected.

Other methods of evaluation include pre- and post-tests which can be administered before and after the CME activity to determine knowledge gain or attitude changes. Unfortunately, these do not measure whether new knowledge will be applied to the physician's practice.

Data from collected evaluations provide useful information for planning future CME activities. This information can be discussed during the periodic evaluation (yearly) or the overall CME program. Other issues to be discussed at the overall review would include the budget, operational aspects, program planning and whether the CME program is a reflection of the mission statement.

ESSENTIAL #6
PROGRAM
ADMINISTRATION
The sponsor shall provide evidence that management procedures and other necessary resources are available and effectively used to fulfill its continuing medical education mission. The sponsor shall:

1) Document an organizational structure for CME and its administration, designating an entity responsible for CME and delineating its authority.

2) Identify responsible individuals who will maintain continuity of administration.

3) Describe an internal review and control procedure, including budgetary practices, to ensure effective utilization of resources in fulfilling the CME mission.

4) Provide a budget for the overall CME program and its major components.

5) Utilize component faculty.

6) Provide appropriate facilities for CME programs.

7) Have mechanisms to record and, when authorized by the participating physician, to verify participation.

A designated or part-time individual (CME Chairman) should be the central figure and decision maker for the CME program. An active committee, consisting primarily of physicians, should meet regularly to discuss CME issues. A committee provides continuity in program planning and administration, assistance to the CME Chairman, and representation of the hospital staff. All discussion at CME committee meetings should be documented in the form of minutes.

A budget, regardless of the size or scope of the CME program, should provide a system of financial accountability and give assurance of the integrity of the CME program. The budget should adequately reflect the objectives of the CME program:

1) Scope of the program
2) Available resources and their costs
3) Funding sources
4) Operational expenses

Faculty should be selected not only for their knowledge of the topic, but for their ability to reach an audience. The appropriate facility for an activity should be determined by the number of participants expected, the method of presentation, and accessibility. The location should complement, not take away from, the activity.

A sufficient mechanism for recording physician attendance should be in place. It is recommended that records be kept for at least six years and made available to participants upon request.

Questions are encouraged and should be addressed to:

Kimberly Allyn
CME Coordinator
Rhode Island Medical Society
106 Francis Street
Providence, RI 02903

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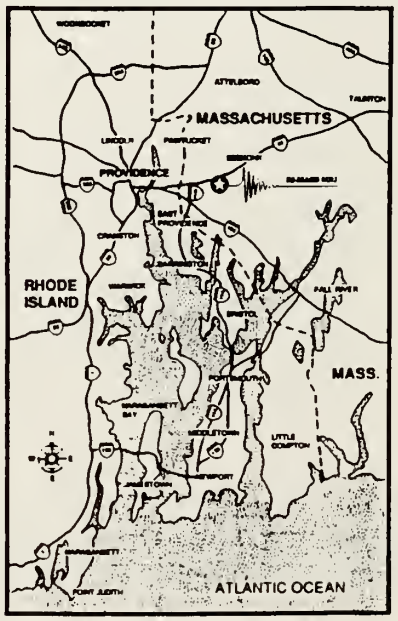

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Am Fam Phys 1987;36:133-140

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Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of non-susceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Hypersensitivity reactions have been reported in about 1.5% of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of **serum-sickness-like** reactions have been reported with the use of Ceclor. These are characterized by findings of erythema multiforme, rashes, and other skin manifestations accompanied by arthritis/arthralgia, with or without fever, and differ from classic serum sickness in that there is infrequently associated lymphadenopathy and proteinuria, no circulating immune complexes, and no evidence to date of sequelae of the reaction. While further investigation is ongoing, **serum-sickness-like** reactions appear to be due to hypersensitivity and more often occur during or following a second (or subsequent) course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults with an overall occurrence ranging from 1 in 200 (0.5%) in one focused trial to 2 in 8,346 (0.024%) in overall clinical trials (with an incidence in children in clinical trials of 0.055%) to 1 in 38,000 (0.003%) in spontaneous event reports. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy; occasionally these reactions have resulted in hospitalization, usually of short duration (median hospitalization = two to three days, based on postmarketing surveillance studies). In those requiring hospitalization, the symptoms have ranged from mild to severe at the time of admission with more of the severe reactions occurring in children. Antihistamines and glucocorticoids appear to enhance resolution of the signs and symptoms. No serious sequelae have been reported.
- Stevens-Johnson syndrome, toxic epidermal necrolysis, and anaphylaxis have been reported rarely. Anaphylaxis may be more common in patients with a history of penicillin allergy.
- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1% and, rarely, thrombocytopenia and reversible interstitial nephritis.

Abnormalities in laboratory results of uncertain etiology:
- Slight elevations in hepatic enzymes.
- Transient lymphocytosis, leukopenia, and, rarely, hemolytic anemia and reversible neutropenia.
- Rare reports of increased prothrombin time with or without clinical bleeding in patients receiving Ceclor and Coumadin concomitantly.
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

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Identification of Patients: Names, initials should not be used. Use of numbers is a preferable form of identification.

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Correspondence: All correspondence relating to publication should be addressed to: Managing Editor, Rhode Island Medical Journal, 106 Francis Street, Providence, RI 02903.